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ARCHIVES OF OPHTHALMOLOGY.

EXPERIMENTAL STUDIES OF TRACHOMA.[1]

Chronic Granular Conjunctivitis Induced in *Macacus rhesus* and Chimpanzee by Inoculation of Cultures of a Microörganism Isolated from Cases of American Indian Trachoma.

By Dr. HIDEYO NOGUCHI, New York.

(From the Laboratories of the Rockefeller Institute for Medical Research.)

(*With six illustrations on Text-Plates XIV. and XV.*)

As a result of the study by bacterial cultures of material derived from five cases of trachoma occurring in pupils in the Albuquerque Indian School, Albuquerque, New Mexico, a microörganism has been isolated which induces in *Macacus rhesus* monkeys and in the chimpanzee a chronic granular conjunctivitis resembling that observed in the patients from whom the microörganism was obtained. The diagnosis in the cases studied was made by Drs. Parlett and Richards, of the U. S. Indian Service, and in all instances scar tissue was present at the time of collection of the material. Re-examination by Dr. Richards one year after the material was collected showed that trachoma was still present.

Direct inoculation of the conjunctiva of four *Macacus rhesus*, a chimpanzee, and an ourangutan, with the trachomatous material failed to induce infection, the animals remaining

[1] Presented before the Section on Ophthalmology, May 16, 1927.

normal during four months of observation. The material was injected subconjunctivally on the upper lid, and at the same time the tarsal conjunctiva was slightly scarified with the needle point of the charged syringe.

Various bacteriological procedures were employed, including several which in recent years have been used successfully in the isolation of highly parasitic organisms. Besides the effective microörganism, which was obtained from four of the five cases, four other kinds of bacteria were isolated from the material studied, including (1) *Staphylococcus albus* and (2) *Bacillus xerosis*, both of which were present in all five cases; (3) a minute Gram-negative, motile, mucin-producing chromogenous bacterium which grows well on ordinary culture media, and which was obtained in all but one case; and (4) a large, sarcina-like organism, sometimes Gram-positive, sometimes Gram-negative, which was isolated from four cases. The microörganism which proved to be active is a minute Gram-negative, pleomorphic bacilliform organism, motile only under certain conditions, which grows on a semi-solid medium containing fresh animal serum and hemoglobin but not on any of the ordinary culture media and only occasionally on blood agar.

Macacus rhesus monkeys were used for testing the pathogenicity of the cultures, the inoculations being made by subconjunctival injection and epithelial scarification. The staphylococcus, which was inoculated into three monkeys, gave rise to an acute purulent conjunctivitis which completely subsided in about ten days without producing chronic inflammation. The inoculation of *B. xerosis* into two monkeys produced no appreciable effect. The sarcinoids were inoculated into twenty monkeys and the motile bacillus into ten. The largest number of animals was used in the case of these two organisms, because they appeared at first to be more unusual and therefore more promising than the others. Both induced a moderate mucopurulent conjunctivitis which lasted about a week, but no follicles developed during several months of observation. Somewhat later five monkeys were inoculated with a mixture of two strains of the Koch-Weeks bacillus; no conjunctivitis and no follicle formation developed. The results of all these tests indicated that none of these five

ILLUSTRATING DR. NOGUCHI'S ARTICLE, "EXPERIMENTAL STUDIES OF TRACHOMA."

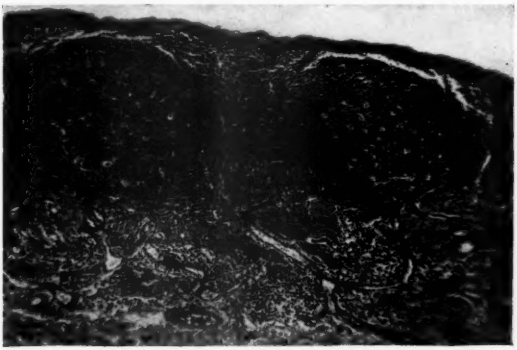

FIG. 1. Trachoma follicle, Fort Defiance. Case 1. X 52.

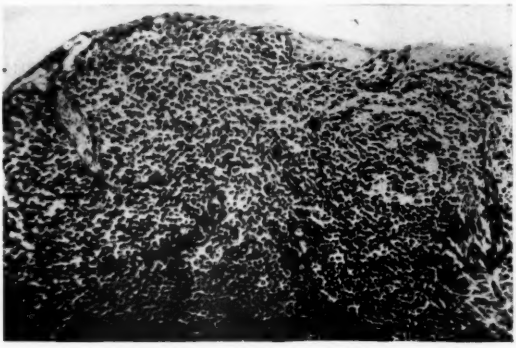

FIG. 2. The same. X 156.

FIG. 3. Less well-defined follicles, Albuquerque. Case 5. Note marked thickening of epithelial layer. X 156.

ILLUSTRATING DR. NOGUCHI'S ARTICLE, "EXPERIMENTAL STUDIES OF TRACHOMA."

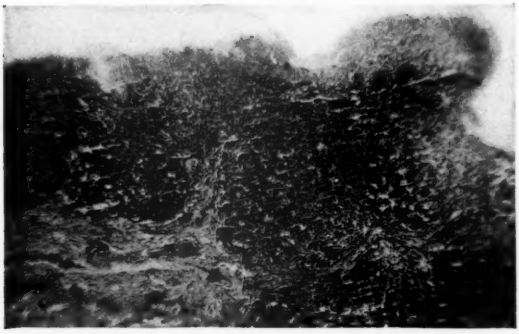

FIG. 4. *M. rhesus* 79, 37 days after inoculation, showing infiltration and follicles. X 52.

FIG. 5. The same. X 156.

FIG. 6. *M. rhesus* 84, 234 days after inoculation, showing sclerotic changes. X 156.

organisms, all of which grow on ordinary culture media, is capable of inducing granular conjunctivitis in *Macacus rhesus* and that the conjunctiva of this animal does not easily react with follicle formation.

The effects were wholly different after the inoculation of the bacillus which grew only on the special media. Twelve monkeys altogether have been inoculated with this organism, of which eight have developed a slowly-progressing chronic granular conjunctivitis resembling trachoma in the localization and character of the lesions. The effects became noticeable in two to four weeks after inoculation, and the lesions gradually extended over the tarsus. In the course of from two to five months the appearance of the inoculated conjunctiva approached more and more that of the human trachomatous conjunctiva in the early stages of the disease; follicle formation and tarsal thickening occurred. In one monkey (*M. rhesus* 84) scar formation in the tarsal conjunctiva became apparent seven months after the inoculation. In several monkeys (Nos. 79, 85, 127) the uninoculated eye soon became involved, owing to spontaneous transmission from the inoculated eye, while in one instance only (No. 84) the condition remained confined to the inoculated eye until experimentally transmitted to the other side.

In three monkeys inoculated with the same culture the lesions developed, remained for a short time, and then receded. In only one monkey was the inoculation entirely negative.

The lesions induced in the conjunctiva of *rhesus* monkeys by means of the cultures were directly transmitted to normal *rhesus* monkeys in several instances. In four of the monkeys to which direct transfer was made the lesions are still progressing, in two they have remained stationary, and in three they have receded. In three animals the direct inoculation of material from culture monkeys failed. The character and localization of the follicular lesions in the four passage monkeys were identical with those in the monkeys successfully inoculated with cultures.

From the upper eyelids of one of the passage monkeys transfer was made to two *rhesus* monkeys and two chimpanzees. One of the *rhesus* monkeys developed very mild lesions, the other escaped infection. One of the chimpanzees, however,

developed within four weeks a chronic granular conjunctivitis which has steadily progressed. For four months the lesion was confined to the inoculated (left) eye, but at the end of that time the other eye also began to show the granular condition. The granules are grayish, succulent, and contiguous with one another, occupying the fornices, and nearly half of the tarsal conjunctiva. The conjunctiva of the lower lid is also studded with similar follicles. There is general papillary hyperplasia and a slight mucopurulent secretion. The other chimpanzee inoculated at the same time showed only a transient reaction.

A small portion of the lesion removed from the chimpanzee forty-nine days after inoculation gave rise to characteristic lesions in a *rhesus* monkey. The transmission of the disease from *rhesus* to chimpanzee and back to the *rhesus* monkey clearly shows the communicability of the condition.

The bacillus requiring special conditions for cultivation has been recovered from *rhesus* monkeys on two occasions and also from the chimpanzee. It is, however, difficult to isolate, since it does not grow on plate cultures, necessitating that the material used for inoculation must be relatively free from contaminating organisms.

The bacillus has occasionally been demonstrated in sections and films derived from human and monkey materials.

In microscopic characters the lesions produced in *Macacus rhesus* closely resemble those of human trachoma. The only striking difference arises from the fact that the conjunctiva of the monkeys possesses no adenoid layer between the epithelium and the tarsus and no papillæ comparable with those in the human conjunctiva; hence in this species the papillary hyperplasia of the tarsus is absent. Nevertheless, wherever there occurs in the monkey eyelid sufficient loose subconjunctival tissue there arise typical lymphoid infiltration and follicle formation, as shown in the regions near the fornix. The epithelial and subepithelial layers of the monkey eyelid over the tarsus often show a marked infiltration, though it is not of the papillary type.

The characteristic appearances of the follicle—the darker-staining small lymphocytes in a laminated periphery and a lighter center of large vesicular mononuclear cells, often capsulated—agree in human and monkey lesions. In both the

follicles contain the so-called Leber's kōrperchenzellen or Villard's phagocytes, and mitosis is common in the central area.

In the *rhesus* monkey (No. 84) in which the scar tissue was present, the lymphoid infiltration and follicles had almost disappeared at the time of the animal's death from tuberculosis seven and one-half months after inoculation. The deformed tarsi were covered with thickened epithelium, and a degree of entropion existed.

The disease known as trachoma among the American Indians has all the characteristics of classical trachoma and leads to the same disastrous results. It is a chronic granular conjunctivitis which ends in deformity of the lids through sclerosis of the mucosa and tarsi and impairment of vision through pannus. Trachoma has been transmitted directly from man to chimpanzee and lower monkeys by previous investigators (Nicolle, Morax, Hess and Rōmer, and others), but so far as can be ascertained from their publications pannus was not observed among these animals. No one has produced a similar result with pure bacteria. The culture obtained in the present investigation produces on inoculation in monkeys the essential clinical and pathological changes described in the direct transmission experiments. That failure followed the direct inoculation, and yet positive results were secured from the cultures, may be explained by the fact that the original material contained too few bacilli to break down the natural resistance of the conjunctival tissues, while the large numbers of bacilli in culture accomplished this effect. The similar instance of Oroya fever may be cited, in which a rich culture of *Bartonella bacilliformis* is capable of inducing infection of monkeys, whereas the original blood, from which the cultures were obtained, is unable to do so.

ON THE MECHANICS OF THE SQUINT OPERATION.[1]

By Dr. HENNING RÖNNE, Copenhagen.

WHILE there are few subjects which have been treated so often as the technique of squint operation, in particular the possibilities of placing the sutures in advancements, the physio-mechanical conditions which determine the correction of the eye has, to a peculiar degree, been left out of consideration. By no means can it be said that there is here a question of obvious facts or that they are equally without importance with regard to our operative practice. On the contrary, many of the authors who have written about technique, choice of operation, and indications of the latter, reckon with obviously erroneous points of view.

I shall start with the most simple mechanical condition in the operation, namely, how far the correction depends on the change of the muscle-insertion, which many years ago was quite correctly demonstrated by Krenchel.

If, by an advancement or a tenotomy, we shift the point of insertion of the muscle 10*mm* on the surface of the sclera, by what amount will then the eyeball be turned?

I think most persons will immediately assume that the apex of the cornea will move a corresponding amount. But a simple consideration will show that this assumption is entirely false.

If an advancement and a tenotomy of 10*mm* each are performed on the same eye it is evident that the total turning effect must be 10*mm*. The result of the single operation, therefore, must be one half of the amount of advancement or recession.

[1] Read before Eye Section, N. Y. Acad. Med., May, 1927.

After a tenotomy of the tendon of a muscle, the muscle—owing to its tension—will retract but little into the orbit and will, in consequence of its shortening, have a lesser tension than its opponent. The equilibrium has thereby been disturbed, the opponent will, of course, contract so much that the tension of the two muscles becomes equal and equilibrium is thereby reëstablished.

As the opponent is still attached to the eye, the corrective turn will be equal to the shortening of the opponent. As the eyeball retains its position in the orbit, the total length of the loop formed by the muscle and its opponent will remain unaltered, and both muscles together will then have shortened by an amount equal to the "amount of recession." As the two muscles are of about the same length both will shorten to the same degree. By the globe following the opponent the amount of correction only becomes as much as one half of the "amount of recession," correspondingly the correction in advancement will correspond to one half of the amount of advancement.

After tenotomy, therefore, the two corresponding muscles will be shortened and relaxed, and this will entail an insufficiency in their action, but this insufficiency will apply to both to the same degree, and is thereby already compensated for to a certain extent. A one-sided weakening of the muscle operated, or vice-versa, a strengthening of it by advancement, can therefore not occur. As we now after internus tenotomies often observe such an insufficiency of the operated muscle only, we can arrive at the conclusion that this insufficiency cannot be due to the weakening of the muscle as a result of the decreased tension, but must be due to a technical failure in the union of the tendon to the globe; it is therefore a matter of an insertion insufficiency, and not of a contraction insufficiency.

But the operation causes at first a contraction insufficiency of both muscles in the loop of muscles under treatment, even if it is sometimes veiled by an insertion insufficiency of the tenotomized muscle.

We shall obtain more information on this matter if we examine the muscle conditions in exophthalmos where there occurs a permanent lengthening of all straight muscles and a corresponding shortening of the oblique ones. Experience

shows the almost incomprehensible fact that no coördinative disturbances arise in the complicated coöperation of the eye muscles, even in cases of a considerable, slowly developing exophthalmos. This can have no cause other than that the muscles very quickly adapt themselves to a new length and, therefore, do not behave as pathologically shortened or lengthened muscles but as normal muscles. This fully agrees with what we otherwise know about the behavior of muscles. Every muscle has an optimal contraction length at which the work performed is at its maximum and the thermic or energy loss at its minimum. A muscle which has shortened or lengthened will, when working, have a mean contraction length different from its optimal contraction, and will therefore naturally adapt itself accordingly. I can give you a quite striking example of a change in length of a muscle. In the Danish Colony, Greenland, the Europeans walk about in heelless footwear. But the change from heeled footwear to heelless, and vice versa, causes for a time pains in the calves which proves that the muscles physiologically can and must adapt themselves to a new length. I therefore believe that the muscles of the eye after the change of the length by operation behave like normal muscles and that there does not exist any contraction insufficiency. The postoperation insufficiency may arise from an elastic union of the tendon to the globe.

The fact we have just discussed, the adaptation of the muscles to a new length, is undoubtedly of importance with squint pathogenesis. Although this question has for many years been treated in ophthalmological journals with silence, one surely remembers the time when discussion on this subject was lively, when the dispute existed between the opinion presented by Albrecht von Græfe that squint was due to a muscular anomaly, a pathological contraction of the internal muscle, and the assumption by Donders of an abnormal innervation as the cause of squint. It may be said that the dispute died out as a consequence of the fact being recognized that neither of these two theories by themselves was sufficient to explain the clinical state of squint.

It seems to me that the opinion of a change in the length of the eye muscles renders the origin of squint comprehensible

in a quite natural manner. It must be assumed that squint arises as an innervation anomaly, that is, what could physically be called a dynamic squint. The resulting active shortening of the muscle must have the tendency to pass over into a passive shortening as the average amount of the muscle contraction does not coincide with its optimum, or in other words, dynamic squint passes over into a static one, which is evident by the fact that the squint does not cease when the abnormal increase of innervation is discontinued by correction of the hypermetropia.

After this digression we shall return to the postoperative insertion insufficiency of an eye-muscle after tenotomy.

I believe that many ophthalmologists today regard this insufficiency as the cause of the most important inconvenience at internus tenotomy, the secondary divergent squint. The old belief still exists that an operation on small children for convergent squint results in a risk of secondary deviating squint, so that the operation is always postponed until the school age, or still later. This postponement is chiefly based on the opinion that secondary squint is due to a spontaneous curing of the muscle anomaly which was assumed to have caused the squint (see Fuchs and Axenfeld's text-books) whereby the original operative correction becomes an overcorrection. But thus assumption seems to make the insufficiency of insertion useless. The question of the pathogenesis of the secondary squint has, therefore, not lost its interest. In a paper published in 1911 I tried to solve the question by statistical means. I went through the vast material of operations from the ophthalmological clinic conducted during fifty years by Grut and Bjerrum, counting the cases of single tenotomy (total 2610) and double tenotomy (1076) in the four decades 1870–1910 and examining how many of these patients had subsequently visited the clinic for secondary squint. I succeeded in finding altogether 112 such patients.

As it appears from the table, the cases of double tenotomy give just twice as great a percentage of secondary squint as those of single tenotomy.

The low number of cases of secondary squint in the decade 1900–09 is naturally due to the fact that only the fewest of

these had been reëxamined, as the examinations ceased with the year 1909.

	Single tenotomy	*Among these secondary squint*	*Double tenotomy*	*Among these secondary squint*
1870–79	374	15	412	36
1880–89	527	13	228	12
1890–99	833	15	256	10
1900–09	876	4	180	0

	Percentage of Secondary Squint	
	After single tenotomy	*After double tenotomy*
1870–79	4 per cent	8.1 per cent
1880–89	2.5 " "	5.3 " "
1890–99	1.8 " "	3.9 " "
Mean	2.8 per cent	5.8 per cent

Furthermore, it may be seen from the figures that the percentual frequency of both secondary squint and double tenotomy decrease during the decades, both indicating the greater precaution taken at the operation in the course of years. Merely statistically this double movement results in its being impossible to take the percentage of the total, as the cases of double tenotomy would thereby get too high a percentage of secondary squint (6.5% against 2.5% after single tenotomy). It was therefore necessary to divide the material according to decades. Of course, the same movement is observable within the individual decades, even if with a smaller influence; it must, therefore, be expected that in the individual decades the percentage of secondary squint will be a trifle too high.

These statistical figures tell their own tale. A double tenotomy will give a tendency to secondary squint twice as great as a single tenotomy. This can mean only that the cause

of secondary squint is a technical failure at the individual operation in the form of an insufficiency of insertion.

In my investigation I compared the conditions of all the cases of squint with those of the secondary squint. Refraction, age at the operation, angle of squint, alternation and periodicity were the same in the primary and the secondary squint. The pathogenesis of the secondary squint is therefore independent of these conditions.

I now want to consider more closely the importance of the age at the time of operation on secondary squint, as it has a direct importance on the indications for the operation.

In the diagram (Fig. 1.) the age at operation is given for all cases of secondary squint compared with the age at operation for all internus tenotomies performed. It is evident that the two curves follow each other closely, and from this it appears that the age at operation is without any importance on the development of secondary divergence. Therefore, far from showing that an operation at an early age should result in frequent secondary squint, the diagram shows the contrary to be the case, which is probably due to the fact that special care had been taken in operating on very small children as there is a general dislike against operations on patients at an early age. There is, therefore, no reason for a postponement of the operation to a later age.

In my opinion, therefore, neither tenotomy nor advancement in itself will reduce or increase the movability of the eye beyond the normal limits. In case of technique failure, an internus tenotomy will result in an insertion insufficiency, which will be the direct and only cause for a secondary divergence.

At an advancement combined with tenotomy the mechanical problems proceed in a somewhat different manner. A very great number of methods have been suggested to secure the advancement sutures in the tendon of the muscle, but the attachment of the sutures to the eye has apparently interested the experimentalists to a lesser degree. In my opinion however, this is the only field where variations in the operative technique really will give effective difference in the mechanics of the operation.

There are two different methods of attachment of the

advanced muscle: (1) The advancement sutures are placed in an exactly horizontal direction through the original muscle insertion or episclerally in the tight subconjunctival tissues and the conjunctiva. It is thereby sought to obtain a good and unshiftable union to the sclera. (2) The needle is passed through the episclera and conjunctiva to a point above and below the center of the cornea. It is thereby sought to place the point of union as far centrally as possible, so as to move the remaining tendon insertion well forward.

The difference between the two methods does, however, not lie in the manner in which the tendon advanced is attached to the eyeball, but in the reaction thereby produced on the previously performed tenotomy. It is quite clear that the advancement must have an effect on the tenotomy. The tension in the loop of muscles formed by the internus and the externus must increase with the shortening of the externus, and thereby a mechanical relation is introduced which must result in an increase of the recession space (the tenotomy-diastasis) and thereby also an increase of the total effect of the operation. In view of this argument, the effect of the combined operation will thus be greater than the total of the separate effects of the two operations together. If the advancement sutures are allowed to encompass a considerable piece of the conjunctiva of the globe beside the cornea there will, however, arise an opposite mechanical moment which, in my opinion, has not been given sufficient consideration. The conjunctiva of the globe will with a considerable force be pulled over in the direction of the muscle advanced and this pull will be transferred to the adjoining part of the conjunctiva where it will appear by the closing of the incision wound for the tenotomy, even when no conjunctival suture has been made. The pull will naturally also be transferred further to the tenotomized tendon itself through the ligaments which connect it to the conjunctiva, and the tendon will again be pulled forward towards its former point of insertion reducing the amount of recession and the effect of the operation being thus lessened. If the advancement sutures lie in the neighborhood of the former point of insertion of the muscles there can not arise any pull in the conjunctiva on the other side of the cornea.

Systematic experiments have shown that the difference

between these two methods are so great that they may almost be termed two entirely different operations. The difference will appear during the operation by the tenotomy wound in the conjunctiva being drawn together and by folds being formed in the conjunctiva of the eyeball over the upper and lower edge of the cornea in case of a distinct conjunctival union of the sutures. In this practice the postoperative pain is greater, because the tension in the loop of muscles is increased while at the same time the amount of recession is reduced. The turning effect of the operation is distinctly smaller but, on the other hand, an instant postoperative insufficiency of the internus is rare while, with the other practice, in my hands, and at first, it was even frequent. It has, however, never caused me more inconvenience than that four to six days after the operation I had to lay a suture in the insufficient internus. This always removed the insufficiency and this method is preferable to making a correction at a later time.

The difference of the attachment of the sutures to the eyeball will also be noticeable in quite a different direction, namely its effect upon the elevation of the visual axis produced by an unsymmetrical position of the two advancement sutures.

The practical significance of this question is evident from the well-known facts that an ill considered insertion of the sutures may result in an undesirable postoperative difference in elevation, and also, that in given cases it may be desirable by the advancement operation simultaneously to correct an already existing deviation in elevation. The significance of unsymmetrical placing of the sutures has been but sparsely dealt with in handbooks on ophthalmic surgery, for instance, not at all in W. Czermak: *Die augenärtzlichen Operationen*, II Aufl., 1908, and only very slightly in Ch. Worth: *Squint*, London, 1921, p. 207; these relations have not received the attention that they merit.

Theoretical consideration as to the effect of the two sutures are not of great importance. For the most obvious supposition would be that the insertion of the muscle would become displaced in the direction of the most effective suture and the mechanical consequence of this displacement would be that the muscle henceforth will not act merely as an abductor

or adductor, but also move the eye in the vertical direction.

An upward displacement of the tendon should then produce a partial upward rotating effect and one would expect that an overacting superior suture would produce a sursumvergence and conversely, that an overacting inferior suture would lead to deorsumvergence. Experiments, however, prove the case to be the exact reverse, for an advantageously placed superior suture moves the eye downward.

If the sutures in the globe are laid at the level of the limit of the tendon insertion a difference in the tightening of the two sutures will not produce any material effect. The theoretically probable consequence will be only an obliqueness of the line of the tendon insertion, to which hardly any mechanical effect can be ascribed. If, however, one of the sutures is laid in the conjunctiva above or below the cornea, a tightening fold will form in the conjunctiva above the edge of the cornea. As this will exert a vertical pressure on the cornea in front of the point of rotation of the eye, it will rotate the top of the cornea in the opposite direction.

This effect may be directly observed in an ordinary advancement by removing the advancement forceps before knotting the advancement sutures. Tieing the upper suture will then give the eyeball a deorsumvergence (downward rotation) which is corrected by the other. It is apparent from the foregoing that it is a question of a purely conjunctival effect, the insertion point of the tendon of the muscle being without significance. In practice it is comparatively easy to correct a pre-existing deviation in the elevation of the visual axis by producing with the first advancement suture an over-correction which is afterwards again corrected by the appropriate position and tension of the second suture.

That the mechanical effect will have an entirely or partly permanent result is probably the consequence of the ability of the muscles to adapt themselves to a given position.

I have in the above tried to regard the conditions of squint operations from a purely mechanical point of view, which I believe is not general.

Of course, the biological conditions, particularly here where an operation with a purely mechanical object is dealt with, are so complicated that a criticism of details can always be raised.

But I hope, at all events, to have pointed out problems which are not quite without importance for any practice in this, perhaps the most frequent of all, operation.

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ROD-PALPATORY METHOD FOR THE EXPLORATION OF SOME OF THE ORBITAL CONTENTS AND OF THE SURFACE OF THE EYEBALL.

By Dr. MARK J. SCHOENBERG, New York.

A DILIGENT search through the pages of text-books on Pathology and one's personal experience bring forcefully to the fore the fact that no tissue in the human organism is spared by disease and senility. There seems to be no exception to this law. This proposition leads us to accept—a priori—in our own field of endeavor the existence of pathological processes occurring in tissues in and around the eyes—of which no mention is being made in text-books on Ophthalmology or Ophthalmic literature.

One could very appropriately ask:

I.

How often has the reader made the diagnosis of a myositis of one or of several ocular muscles? How many times did he come across a synovitis of the pulley of the superior oblique? When did he say to himself that he is dealing with an asthenopia, due to a neuritis of the ciliary nerves? or with a patch of sluggish inflammation in Tenon's capsule? with an epiphora reflexly due to a latent, chronic inflammatory process in the orbit? with some pathologic condition in the tendons of the ocular muscles? With episcleritis situated way back? or with a small patch of periostitis of the orbital walls, further back than our small finger can reach? with a neuritis of one of several ciliary nerves?

Text-books on Ophthalmology are entirely reticent about these conditions and Ophthalmic Journals publish no papers discussing such possibilities.

And yet—these conditions must, occasionally, occur. Why—of all the muscles of the body—should the ocular muscles be always spared? Why the tendons? Why the synovia of the pulley of the superior oblique? Why should the various pathological processes involving the sclera and episclera be always localized in the anterior portion of the eyeball, and only very rarely further back. And the nerve supply of the ciliary body—mostly the long ciliary nerves, why should not they become afflicted once in a while. A priori this all must happen. Is there a way of making a diagnosis?

The purpose of this paper is to call the attention of the readers to a simple and practical method of examination, enabling one to explore in the orbital tissues, on the orbital walls and on the external surface of the eyeball conditions that have neither been described, not even mentioned heretofore in the Ophthalmic literature. The method is a modified form of palpation, using an ordinary slender glass rod (like the one used for applying ointment in the conjunctival sac) instead of the finger, to feel the consistency, thickenings, swellings, hardenings, deposits in the orbital walls, in the tissues surrounding the eyeballs and the outer surface of the eyeball, also to discover tender areas in these parts. The reason for using a rod instead of one's finger is that a rod is so much thinner, it can palpate a smaller area, and it can be pushed very much deeper in the orbit exploring parts entirely inaccessible to the tip of a finger.

II.

The Method. There are two ways of rod-palpating: the direct and the indirect.

By the indirect method we feel the orbital borders, the walls of the eyeball and other orbital contents through the thickness of the lids. By the direct method we palpate with the rod, directly the surface and the walls of the eyeball, without the intermediary of the lids.

How to use the indirect rod-palpation.

First. The patient is told to look up. The glass rod, held like a pen, is applied parallel with, and as near as possible to

the middle of the margin of the lower lid. By using gentle pressure the lid is slowly pushed as far down as possible gliding it over the eyeball: while pushing the lid down, slight pressure is exerted with the end of the rod against the eyeball, first in the course of the inferior rectus, then on each side of it and finally, straight back and upwards, towards the eyeball and downwards towards the orbital wall. In this manner the tendon and part of the inferior rectus muscle, the lower wall of the eyeball below the limbus, the Tenon's Capsule and also the lower wall of the bony orbit are rod-palpated.

Second. The patient is told to look down and the end of the rod is applied on the middle of the upper lid very near its edge and parallel with it. Using slight pressure the lid is slowly glided upward, as far as it goes. The sclera above the limbus, the tendon and superior rectus muscle are being "palpated." After this the rod is directed up and backwards and pressure exerted towards the deeper tissues, mainly the Tenon capsule. The same procedure is used in "palpating" the eyeball to the nasal and temporal side of the superior rectus. Pressure directed up and backwards brings the rod in contact with the superior orbital wall.

Third. To palpate the tendon of the superior oblique and the region of its pulley with its synovial membrane, the rod is applied—on the nasal side of the upper lid and gentle pressure directed up, in and backwards, towards that zone.

Fourth. To palpate the lachrimal gland, the rod is applied on the upper lid, while the eye is looking down and nasally and pressure is directed up—and outward—in the region of this gland.

Fifth. The nasal and temporal side of the eyeball "palpated" with some difficulty by directing the patient to look downward temporally, or down and nasally and applying gentle pressure through the lids.

How to use the direct rod palpation.

First. Anæsthetize the cornea and conjunctiva with a 1% solution of buttin or holocain.

Second. Apply the end of the rod on the zone corresponding to the root of the iris and ciliary body and press gently.

This "palpation" should be applied over the entire ciliary zone, all around the cornea.

Third. Rod-palpate (by applying the end of the rod directly on the eyeball) the upper part of the globe, including the line of tendinous insertion, the tendon and superior rectus muscle: also the Tenon capsule above. (Patient looks as far down, as possible, while the lid is held back with the thumb of the other hand.)

Fourth. Rod-palpate the lower part of the globe, the tendinous insertion, the tendon and the inferior rectus muscle: also the Tenon capsule below. (Patient looks upwards and the lower lid is depressed.)

Fifth. Rod-palpate the nasal side of the globe, including the internal rectus, its tendon and global insertion: also the caruncle and the region of the lachrimal sac. (The patient looks to the temporal side.)

Sixth. Rod-palpate the temporal side of the eyeball, the external rectus with its tendon and line of insertion, also the lachrimal gland region. (The patient looks to the nasal side.)

This method of examination, though very simple and very little time consuming, should be used by those, who try to familiarize themselves with it, first on adults of a more quiet disposition. Children, as it is well known, are not easily manageable and it takes some experience and a good deal of tact to rod-palpate without setting up a defensive reaction on their part—consisting of a complete lack of coöperation, blepharospasm reflex tearing and so on.

The secret of a successful examination of this kind lies in the care and gentleness employed. Only experience gained by the examination of a few hundred patients will tell how much pressure is necessary to obtain the proper information as to actual tenderness or pain, the feeling of swelling, induration, thickenings, rigidity, etc., in the tissues palpated. Often when in doubt, the examination has to be repeated on other days and findings recorded. The comparison of findings between both eyes is frequently very helpful. At times the findings of the first examination—mostly as to tender zones are not found at subsequent examinations. It is therefore important to repeat the examinations at various intervals and consider as positive

only those findings, which are constantly present; while those, that are only occasionally present should be recorded only as worthy to be watched without forcing any definite conclusion.

It is necessary to know how a normal individual reacts to rod-palpation in order that we have a standard to go by when we meet with a pathological case.

A. Many patients will accuse a sharp pain like that caused by the prick of a needle when the rod is pressed more deeply towards the Tenon capsule. This seems to be due to pressure on a nerve filament, otherwise, it could not be compared with the prick of a needle.

B. Some patients lachrimate profusely—and this occurs mostly when the pressure is exerted in zones not identified with that of the lacrimal gland. It is not clear yet whether this is to be considered as normal or abnormal reflex. Possibly when the lachrimation is very profuse, it may be regarded as a pathological manifestation.

C. At times, the moment the rod-palpation is applied above, the eye which is supposed to look down turns upwards and only repeated admonitions persuaded the patient to control and to look steadily down.

D. Some patients,—mostly young women, are very sensitive about their eyes and orbits and it requires training of the patient and considerable gentleness to get them to relax and to coöperate. In such patients the lids close tightly, the eyes tear and the head moves away as if ready for defense, against a fearsome or apparently harmful examination.

E. Some people call pain, where there is only a sensation of pressure and others minimize their impressions. For both types, the examiner has to be on the look out, so as not to call a case negative, when the opposite is true and vice versa.

III.

What does a routine rod-palpatory exploration reveal, and in what conditions are we apt to find one or more tender areas in the orbit? So far only a few hundred patients have been examined. Their ages range between 5 and 80 years and of both sexes. These patients were the regular run of cases as we meet them

in private practice and dispensary. Surely not less than one out of ten patients presents one or several painful areas in the deep tissues around one or both eyes. Most often patients are not aware of these tender or painful zones. They come with all sorts of vague complaints about their eyes and head, but they never speak of a definite pain, that they can localize in or around their eyes.

In one large group belong the cases with various complaints of asthenopia, not relieved by the most painstaking correction of the error of refraction.

In a second group are some cases with chronic iritis and iridocyclitis, episcleritis and scleritis.

In a third group belong patients with an inflammatory affection of the muscular sheaths, of the muscles themselves or of Tenon's capsule, frequently found in patients with "chronic rheumatism" or fibrositis.

Fourth. Some cases with retrobulbar neuritis, even of long standing.

Fifth. In some cases of epiphora with normal lachrimal passages, in which the tender area is the starting point of a reflex hypersecretion of the lachrimal gland—reflex epiphora.

Sixth. In patients with patches of congestion in the region of the pulley of the superior obliques, due to acute infectious diseases—like grippe, tonsillitis, etc.

Seventh. In ill refracted patients or in some patients with errors of refraction, wearing no glasses at all.

Eighth. In trigeminal neuralgia, various types of hemicrania and migraine.

Ninth. In some cases of more or less active or congestive glaucoma.

Tenth. In some cases of nasal sinusitis, with a transgression of the inflammatory process in the orbital tissue—not necessarily an orbital cellulitis.

Eleventh. In the period of incubation of acute infectious diseases, in which patients complain of headache and pain in the eyeballs—mostly when moving the eyes in various directions or rolling them around.

What may be the actual condition of the tissues involved in these tender areas?

We may assume that we deal either with patches of conges-

tion or inflammation or with a neuritis. The careful analysis of the findings in each case may lead this to the proper conclusion regarding their diagnostic significance.

I repeat: ***the secret of a successful use of the method of rod-palpation lies in the care and gentleness employed.*** Only experience gained by the examination of a few hundred patients will tell how much pressure is necessary to obtain the proper information as to actual tenderness or pain, and as to the presence of swelling, induration, thickenings, rigidity, etc., in the tissues palpated. Often, when in doubt, the examination has to be repeated on other days and the findings recorded at each examination. The comparison of findings between both eyes is frequently very helpful. At times the findings of the first examination are not the same as on subsequent examinations. It is therefore important to repeat the examinations at various intervals and to consider as positive only those findings which are constantly present: while those that are only occasionally present should be recorded as worthy to be watched without forcing any definite conclusion.

It should be added that rod-palpation may be of some therapeutic use when skillfully employed for massaging the deep tissues of the orbit.

X-RAY THERAPY IN CERTAIN TYPES OF GLAUCOMA.

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OVER-RIPE cataracts are often subluxated and sudden increases of tension occur before and not infrequently afterward. The blind eye of glaucoma simplex usually contains a partially cataractous lens and the blood vessels are notoriously weak. Liability to sudden elevations of intraocular tension with blood in the anterior or vitreous chambers is the logical result. Physical effort with the head low is a common part of the history of these acute conditions but many are without a known cause. These eyes present a different problem than the usual cataract or glaucoma because the first and all-important demand is relief of pain and not restoration of vision. Measures not to be thought of, if we aim to save vision, may be legitimately used when an already blind eye is under consideration.

A lady of 70 had been successfully operated for a cataract of the right eye and was entirely satisfied with the vision obtained which was close to normal. The second eye became blind from the same cause but she would not consider treatment of any kind. Three years went by before her return to the clinic with a very hard, painful and red eye. The cataract in the unoperated eye had in the meanwhile passed into the over-ripe stage and the anterior chamber was filled with blood up to the level of the pupil which was somewhat larger than the normal. The finger tension was stony hard but our patient was still unwilling to be operated and would not even enter the hospital. This attack came on after scrubbing the floor. Pilocarpine, eserine and cocaine solution was used without result upon either the tension or the patient's point of view. While

debating what should be done to relieve the severe pain and avoid an enucleation, if possible, there came to mind an article which I had seen somewhere reciting the effects of X-ray therapy in glaucoma hæmorrhagicum. Feeling that no harm could come to this already sadly neglected eye from this treatment, I sent her to Dr. Winfield Perkins who treated her with a mild, stimulating dose of the X-ray with results so strikingly immediate that we were all most favorably impressed. The pain was relieved within a few hours, the blood absorbed slowly and the redness disappeared in about two weeks. The tension to the fingers was in upper limits. She had three X-ray treatments which relieved her for two years before her return with a very red eye, corneal ulcer and hypopyon up to the pupil edge and severe pain. The tension was not affected. From this, she made a slow but uneventful recovery. In the interval between these last two experiences, the lens has luxated, the nucleus has sunk to the bottom of the fluid containing capsule and the upper edge of the Morgagnian cataract crosses the upper third of the enlarged pupil. The ulcer which occasioned the hypopyon was a very superficial central loss of epithelia with a twisted line of infection which seemed to reach the posterior corneal surface. This is quoted to show the condition of the eye and make plain the many aspects of the situation and how dangerous these eyes are. In the meanwhile, search for this article suggesting the treatment was made. It was by Hessberg, *Klin. Monatsblätter für Augenheilkunde*, volume lxiv., p. 607; with the title—"Die Behandlung des Glaucom hæmorrhagicum mit Roentgenstrahlen." 1920.

The next case in which the treatment was applied was the blind eye of glaucoma simplex.

An elderly lady of 70 had been blind in one eye for a year or more and came with the complaint of poor vision in the other and difficult vision at night. The good eye had a vision of $\frac{11}{12}$ with correction and the finger tension of the blind eye was slightly plus. She refused operation and came infrequently until an acute attack in the blind eye without known cause. The eye was stony hard, very red and painful and as she would have no operative procedure upon an eye already blind, it was thought wise to try the effect of the X-ray. The results were as in the first case. About six months later, she had another acute attack coming on right after the tension was taken and which read 18*mm* for the right and 50*mm* for the left eye; Schiotz.

Again the X-rays were used with immediate result. The lens of the blind eye is partially cataractous and the tension now to the fingers is in the upper normal regions. The third patient also had two attacks in an eye blind for some years from glaucoma simplex. The other eye had normal vision with correction and the fields showed but little change. The Schiotz apparatus recorded the tension as 20*mm* for the right and 40*mm* for the blind eye. Without known cause, while using miotics, he came with a very painful, red eye which was immediately referred to Dr. Perkins with very satisfactory results. He had six treatments all together and the tension was 15 and 45*mm* respectively.

Six months later, after working on his hands and knees, reaching under a bath tub, he had a very severe attack of pain with the eye stony hard to the fingers. The X-ray treatment relieved the acute symptoms but the slit-lamp showed corneal œdema, new blood vessels in the iris, cornea dull centrally with a few vacuoles forming just beneath the surface, and as the tension was 22*mm* and 60*mm* respectively and still some pericorneal injection I was able to convince him that a trephine was indicated. This lowered the tension and gave him a white eye with a tension 1 year later of 18*mm* and 25*mm* respectively.

One naturally hesitates to use radium or the X-Ray upon an eye with a good lens or retina; again, one very properly turns to the well indicated treatment which gives results when the vision of an eye is at stake in acute glaucoma. This treatment is not offered as a substitute for methods we know to be reliable in glaucoma simplex or cataract. We do not advise leaving a lens in an eye to become over-ripe, or allowing an eye blind from glaucoma with tension above normal to go without constant consideration and effort to reduce the tension to within reasonable limits. If patients refuse our best advice and appeal for relief of pain with the eye concerned known to be blind, this treatment seems in place. We also have elderly patients and some who are really ill, who might properly be included in the group for whom this might be considered. It is quite natural to ask how the results described are produced. The literature contains nothing concerning glaucoma treatment by the X-ray except the article by Hessberg. Schussler tried raying the adrenals, and the cervical ganglion of the sympathetic has been treated in the same way. Corbett of Boston has reported some success with radium in glaucoma.

Hessberg was led to his treatment of 5 cases of hæmorrhagic glaucoma by the results of the X-ray treatment in controlling uterine hæmorrhages. His results were very satisfactory but one eye was later enucleated. He believes the good results are the result of obliteration of the capillaries, stopping the blood flow, and having also an effect upon the pain and tension. He quotes Gassmann as stating that the main effect of this therapy is upon the blood vessels producing a fiber separation of the elastic coat; degeneration of the intima, vascularity and disappearance of muscularis. At a recent meeting of the Vienna Ophthalmological Society, January, 1927; Sgalitzer said the good effects of the X-ray in brain tumor cases depended not altogether upon the effect upon the tumor itself but particularly upon the inhibition of the secretion of the cerebro-spinal fluid by the choroid plexuses of the ventricles, relieving the internal hydrocephalus. He stated that this inhibitory influence had been proved in dogs and quoted a case with cerebro-spinal fluid dripping from the nose at the rate of 16 drops a minute, for five months. This was relieved in three hours but recurred in 10 days. One week later, it ceased and 6 months later is in the same satisfactory condition. There is some analogy in the functions of the ciliary body and the choroid plexus and it is not far-fetched to suggest that the good results in eye cases may be due not alone to the effect upon the blood vessels but also to the inhibitory influence upon the secreting function of the ciliary body.

It would not seem possible that a result so prompt could be construed to mean that an organic obstruction had been relieved. It would hardly seem possible that anything more than a local swelling of the ciliary body, obstructing the angle of the anterior chamber or a hypersecretion could be relieved so soon.

I have tried this treatment a few times in glaucoma simplex and am in doubt whether it does any good, but have not seen any bad effects. Twice, it has been used in sub-acute glaucoma when patients would not be operated, without result. One of these cases later consented to a trephine and was relieved of all symptoms, and has fair vision.

Perhaps obstruction not due to local congestion, but an organized barrier such as occurs when the iris is applied to the

chamber angle, was the cause and not a hypersecretion, or an obstruction due to swelling only.

It is certain that the result is prompt when obtained and if delayed, the treatment should be abandoned.

It is hoped that cases of increased tension with deposits on the posterior surface of the cornea seen with the slit-lamp, can be treated as they would justify the diagnosis of secretion hypertonia which, if relieved, would corroborate the idea that secretion can be inhibited in the ciliary body as in the brain.

The treatments used by Hessberg are described in his article as follows—"6 Bestrahlungen. 9 bis 20 × 2 bis 4*mm*. A. F." Dr. W. H. Dieffenbach translated this as follows—"6 Radiations. 9 to 20 units of skin exposure. 2 to 4 millimeters of Aluminum Filters."

Ophthalmological literature contains much about the evil effects of radium and X-Rays, also the damage from ultra-violet and the rays of the red end of the spectrum, but the only additional references which are in place here are—Corbett. "Effect of Radium on Glaucoma." *Boston M. & S. Journal*, cxc., p. 1124. Derby. "Radium in Non-Malignant Eye Diseases." *Boston M. & S. Journal*, cxci., p. 20.

THE CULTIVATION OF LENS EPITHELIUM "IN VITRO."[1]

By Dr. DANIEL B. KIRBY, New York.

(With four illustrations on Text-Plate XIV.)

IT was proposed by Dr. John M. Wheeler that an original investigation be conducted into the nature of the cause of senile cataract and the possibility of its prevention and arrest by nonsurgical means.

The literature contains a number of hypotheses as to the genesis of senile cataract and records the various means used in ancient and modern times to offset the formation of, or dissolve the opacity of the crystalline lens.

If such a complex biologic problem were approached from purely a clinical standpoint, and the study based on observations and treatment of patients afflicted with incipient or mature cataract, it was realized that the same difficulties that have conquered other investigators would be confronted and remain unsolved from a scientific standpoint.

It is not too much to expect that some means of influencing the nutrition of the lens may be found. Since the advent of the use of the slit-lamp and microscope, many opacities and

[1] Read before the Eye Section of the N. Y. Acad. Med., April 18, 1927.

The reader is referred to the complete article published in the *Transaction of the American Academy of Ophthalmology and Oto-Laryngology*, 1926, pp. 137–153.

The basis of this report is derived from work conducted by the author as Research Fellow for the American Academy of Ophthalmology and Oto-Laryngology and by virtue of a grant secured from an anonymous donor by Dr. John M. Wheeler. The experiments were carried on by the Department of Ophthalmology in the Departments of Anatomy, Physiology, and Chemistry at the University and Bellevue Hospital Medical College. Acknowledgments are gratefully made to the Professors of these departments and to Drs. Alexis Carrel and A. H. Ebeling for their courtesies.

ILLUSTRATING DR. DANIEL B. KIRBY'S ARTICLE, "THE CULTIVATION OF LENS EPITHELIUM 'IN VITRO.'"

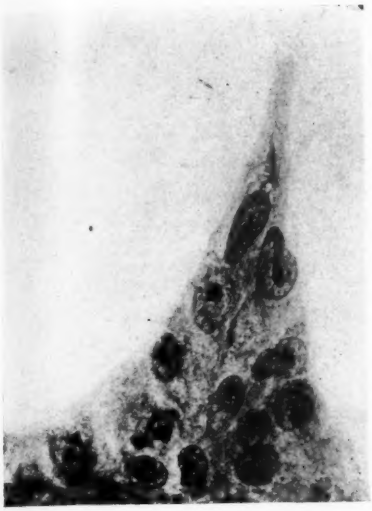

FIG. 1. Fresh explant of lens epithelium. Zenker Acetic. Harris Hæmatoxylin x 220.

FIG. 2. Strain of lens epithelium. Shows mitosis and membrane formation.

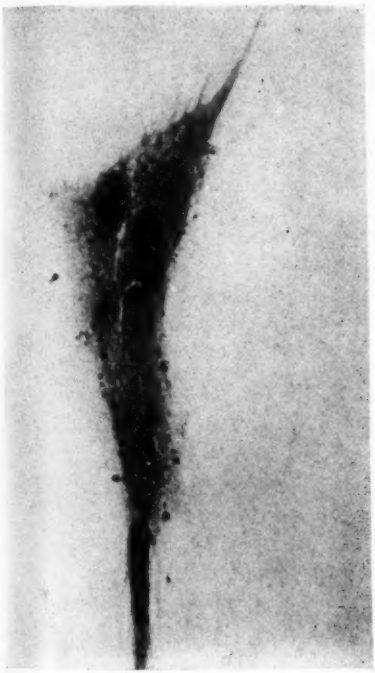

FIG. 3. Cells from strain of lens epithelium. Shows fine processes of protoplasm.

FIG. 4. Cells from strain of lens epithelium. Hernias of the protoplasm.

areas or zones of increased relucency of the lens have been found in apparently normal eyes with normal vision. Covered by transparent cortex they are mute evidence that the lens in point suffered a physiological insult at one time and was able to survive and recover and to bury the opacity by the formation of clear cortex. Cataract may be a deficiency disease and it may be possible to discover the nature of the deficiency.

It was decided to approach the problem from a scientific rather than an empiric standpoint and to go back to fundamentals. A study of the developing lens in the living embryo and of the living cells of the lens offered possibilities. It was also thought that if the cells of the lens could be cultivated outside the body that here would be a new tool, a new standard for our investigation into the nutrition of the lens. If the study of the cause of cataract could be narrowed down to the life and death of the single cell derived from the lens, a great step forward would be taken. The single cell is not so simple, but it is far simpler than the complex lens organ.

Tissue Culture.

Harrison (1) discovered in 1907 that cells could be caused to migrate and divide outside the body.

In 1910, Carrel (2) devised an ingenious technique by which the method of tissue culture is becoming adapted to its main object, (3) the study of the fundamental problems of physiology and pathology, primarily by the isolation of permanent pure strains of cells (4) and by the development of procedures for measuring the rate of growth (5) and for studying the functional activity of the tissue. (6) In 1922, Fischer (7) cultivated the iris epithelium and in 1924–25, Ebeling (8) grew the thyroid epithelium.

In the experiments about to be reported the method and technique of Carrel (2, 3, 4, 5, 6,) were used.

The Growth and Cultivation of the Crystalline Lens Epithelium Outside the Body.

Fischer (7) made numerous explants of the embryo lens with the epithelium of the iris. He reported that the lens tissue did not grow at all.

It appears necessary therefore in describing the cultivation of the lens epithelium outside the body to be able to definitely prove the identity of the tissue explanted.

This will be done *1st* by repeating Fischer's (7) experiments on the iris epithelium to show that if extraneous tissue is explanted with the lens, the extraneous tissue will grow and if the explant of the lens also grows, one will contaminate the other.

2nd—by proving that proper technique of microscopic dissection of the embryo lens frees the lens from all extraneous tissue and

3rd—that the epithelium of the lens so freed and properly cultivated outside the body will migrate and proliferate.

4th—that secondary and tertiary cultures from these can be made so that a strain of pure lens epithelium can be developed.

Experiment No. I.

The lens and vitreous of nine day chick embryos were removed. Usually a ring of pigmented and non-pigmented epithelium of the rim of the optic vesicle also comes away. If this be cultivated in the manner used by Fischer a growth of epithelium from the iris anlage is obtained.

This experiment demonstrates that the iris epithelium can be cultivated and if an unmixed strain of lens epithelium is desired, all extraneous tissue must be removed from the lens. Strains of the iris epithelium were carried along in successive sub-cultures, so that a ready comparison was always available.

Experiment No. II.

After many experiments, it was found that the eyes of chick embryos incubated for five days at 37.5 degrees Centigrade were ideal for dissecting the crystalline lens free from all extraneous tissue. Both prior to this and after it, the protoplasmic adhesions are such as to make it almost impossible to express the lens free from other cells.

After 72 hours incubation of the entire lens it was observed that no growth of cells outside the capsule had occurred.

This experiment demonstrates by cultivation that the lens can be dissected free from extraneous tissue and that the lens

epithelium is contained within a capsule outside of which when uninjured, the cells do not migrate.

Experiment No. III.

Lenses from five day embryos were incubated *in toto* for 24 hours at 37.5 degrees Centigrade. The cultures were then opened, the lenses removed from the media and divided. Outgrowths of cells were obtained from the lens epithelium.

Twenty-four hours is sufficient time for incubation of tissue from this age embryo to exclude any growth of cells extraneous to the cuticula of the lens by incubation.

This experiment demonstrates that it is possible to rule out tissue extraneous to the capsule of the lens by cultivation and then by section of the lens to obtain an unmixed culture of lens epithelium.

Experiment No. IV. Cultivation of Lens Epithelium.

Lenses dissected clean were incised anteriorly and the lens epithelium stripped off easily to the equatorial region, as there exists a potential space at this stage between the lens epithelium and the cortex. The lens epithelium is then explanted on the point of a needle directly to the embryonic tissue juice-plasma clot medium. After 12 to 24 hours the lens epithelium begins to migrate, divide by mitosis and multiply. After 48 hours, observations are recorded. Apparently the entire explant lives, but cells do not wander out from all parts. This depends on the particular conditions of the explant, folding the exposure of the cells, the trauma, and the conditions of the media. Many bizarre shapes have been observed. Small pieces of epithelium do as well as large ones. The explant remains transparent and the newly formed cells are transparent. Protoplasmic processes of various lengths are seen, some being very long.

Experiment No. V.

Extract of the nine day chick embryo eyes was used in place of extract of the whole body. The explants grew well when this was used in place of whole body extract, but were not free from retinal pigment rods in the cells. It was determined therefore to use extract of the embryo body minus the

eyes. This has been done and transparent growths free from any retinal pigment rods obtained. Dr. A. H. Ebeling has been kind enough to examine our cultures and has found on contaminating cells in one produced by our perfected technique.

This experiment demonstrates that lens epithelium can be cultivated in media containing extract from the embryonic eye alone and also from the embryonic body minus the eyes.

Experiment No. VI.

Living explants from the lenses isolated and cultivated as described in Experiment No. IV can be divided and two sub-cultures obtained. These can be in turn sub-divided after cultivation and thus secondary and tertiary cultures obtained. By this means a strain of lens epithelium can be propagated from an original single explant. The divided cultures about double in size every forty-eight hours. The cells are transparent and remain epithelial in type. They do not dedifferentiate.

This experiment demonstrates that lens epithelium cultivated outside the body can be carried on for successive generations. Strain C was carried through seventeen generations over a period of six weeks and was thriving when the work was discontinued, August 9th, 1926. It was then ready for controlled experiments and observations on the rate and character of the growth, the cellular reactions and the substances produced by the cells.

Discussion.

In this study of the cause of cataract and the possibility of its prevention or arrest by non-surgical means, the problem has been approached from the scientific side, namely, an experimental study of the nutrition of the crystalline lens. The first step forward has been taken, that of the cultivation of an unmixed strain of lens epithelium outside the body.

The lens organ grows constantly throughout its normal life. The cells are not cast off but are constantly being compressed and by a process of sclerosis forming the nucleus of the lens. This growth energy of the lens epithelium in life may be expressed as a function of the concentration of the growth

producing and the growth inhibiting substances taken up by the lens. There must be a delicate balance between the two factors to maintain the normal anatomic relations. Tissues in the living body may continuously receive substances in very high dilutions. It may or may not be possible to detect the presence of such substances in experiments outside the body. Can it be that there is such a substance which has to be in a *specific* manner with the growth and differentiation of the crystalline lens throughout its normal life? If so, is this substance an intraocular or an extraocular product?

The transparency of the tissue of the lens is brought about by a certain physico-chemical combination of protoplasm that changes the index of refraction of the cells. The secret or principle of this is contained within the living transparent cells of the lens waiting to be discovered. These cells have a peculiar composition such as is found no where else in the body. The osmotic coefficient is different, the water-binding power of this protein is unique. Are the earliest signs of incipient cataract as observed with the aid of the slit-lamp and microscope, namely, lamina separation, formation of globules and vacuoles, water-splitting of fissures, but an indication of changes in the protein of the lens affecting its water-binding power?

These are some of the questions to which an answer must be found before progress can be made in the study of the nutrition of the lens and the cause of senile cataract.

Conclusions.

1. It is possible to dissect out the lens of the five day embryo chick in such a manner as to free it from all extraneous cells.

2. Lens epithelium even at a very early stage is contained within a capsule.

3. Lens epithelium under proper condition will live, migrate, divide, and multiply outside the body in primary explants.

4. An unmixed strain of lens epithelium can be propagated from a primary explant by successive sub-cultures. This strain can be utilized for controlled experiments on the nutrition of the lens.

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THE CAUSE OF THE GLAUCOMA OF HYPERMATURE CATARACT.

By Dr. H. GIFFORD, Omaha, Nebr.

IN 1900 (*Am. Journ. of Oph.*, xvii, 289) I contended, in opposition to the previously accepted view, that the glaucoma so often noted in connection with the spontaneous "cure" of cataract, is not the cause, but the effect, of the lens disintegration which restores useful sight to some of these patients. At that time, I was inclined to believe that the glaucoma was the result of mechanical irritation from the loose nucleus; plus, perhaps, an obstruction of the outlets by lens debris. In my second article (*Am. Journ. of Oph.*, 1918, 83) I expressed the view that chemical irritation from the lens-disintegration of hypermaturity was the main cause of the glaucoma. This explanation is rejected by Arnold Knapp in his article on Glaucoma in Morgagnian Cataract (these Archives, March, 1927, 124). From his observation of two cases he feels, on the contrary, that the displaced nucleus must cause some mechanical irritation of the ciliary processes and thus produce glaucoma. He asks, "If the absorption of the cortex is the toxic agent, why in these two cases, did not symptoms develop before the cortex was absorbed, and if the liquified cortex be phlogogenic why do we not see more frequently any reaction on the part of the iris and ciliary body; and why is the glaucoma always the important complication?" "Furthermore, if the fluid cortex were toxic why should it not at least cause a few synechiæ?" The answer to part of these objecting questions is very neatly given in the history of Knapp's second case. Here with hypermaturity, but before the Morgagnian cataract had developed (i.e. before the nucleus had been deprived of its cortical support), the eye became red

and painful, the tension rose to 44, and a single corneal deposit with a few slight synechiæ formed. Better evidence that in the pre-Morgagnian stage of hypermaturity, irritating products are produced which cause glaucoma, without the mechanical effect of a loose nucleus, could not be asked. Another instance of tension developing in an eye with hypermature but not Morgagnian cataract is given in Case IV of my second paper; and I have seen two others within the last two years. In the latest of these, a woman with the first signs of hypermaturity, in the shape of irregular whitish masses in the anterior cortex, with an almost full-sized, firmly fixed lens, showed quite a red, but not a painful eye and tension of 35, which went down under paracentesis and salicylate to 17.

The fact that the tension in Knapp's patient gradually went down and then in the course of three months, during which the lens cortex nearly all absorbed, went up again to 38, without noticeable renewal of the inflammation, need not imply that the second rise was due to the loosening of the nucleus. This eye was out of observation for three months before it was found to have a renewed high tension and absorption of the cortex. It is not known how long either of these factors had existed. Cases four and six, in my second paper, indicate strongly that a quiet glaucoma from the slow absorption of cortex may occur and persist for months before the nucleus is left bare, and in Case I the remaining nucleus which kept up the painful but non-inflammatory glaucoma, though quite movable, was so attached to the capsule that it did not come into contact with the ciliary processes or iris at any time, and was so small that any drag on these processes must have been insignificant. It seems much more likely that the symptoms were due to the slow absorption of nuclear substance rather than to any mechanical effect. Other cases have convinced me that nuclear absorption can be fully as irritating and tension provoking as the absorption of cortical debris, and that, like the latter, the symptoms may be either obscure or violent.

Knapp's experience, contrary to that of Elschnig, indicates that milky cortex, released by rupturing the capsule during removal of cataract, is not apt to cause irritation and evidently thinks that this speaks against its having any decided toxicity. I too have seen no irritation from milky cortex freed by capsu-

lotomy at a cataract operation, but this is easily explained by the thorough emptying of the chamber, even if it is not washed out.

Furthermore, let me ask what reason there is for thinking that the nucleus, left after the cortex of a Morgagnian cataract has been absorbed, is capable of producing any more mechanical irritation, either by bumping or by traction on the ciliary processes, than a normal lens or a full sized cataract? As long as it stays in the capsule and the latter retains its attachments to the zonule, the nucleus certainly can not come into contact with the processes, no matter how quickly the eye may be moved; hence there can be no "pounding" on the processes; and, as the traction on the zonula from the lag of the lens in eye movements, is directly proportional to its mass, the traction produced by such small remnants of the nucleus as we generally see must be relatively insignificant, even if we allow for the greater mobility which such small nuclei sometimes (but by no means always) show. Finally, in addition to the fact that it has never been shown that abnormal impacts or traction, even if they occurred in Morgagnian cataract, would produce rise of tension; we have the evidence given by congenitally dislocated lenses to the contrary. In spite of the great mobility of such lenses and their great mass, as compared with the Morgagnian nuclei, they notoriously cause no trouble so long as they do not come partly or wholly into the anterior chamber nor fall back into the vitreous.

To sum up, I should say that while the study of my cases and those of Dr. Knapp show plain clinical evidence of the production of increased tension by the products of lens-hypermaturity before there is any increased mobility of the lens or its nucleus, the supposition that such mobility ever causes high tension is without proof or probability.

HOW PRIMARY GLAUCOMA MAY ARISE FROM DISTURBANCES IN THE PHYSICO-CHEMICAL FORCES WHICH REGULATE INTRAOCULAR FLUID EXCHANGE.[1]

By Dr. J. HERBERT WAITE, Boston, Mass.

Summary

(With two figures in the text.)

The ocular capillary walls and the ciliary epithelium, like other animal membranes, allow the free passage of water and of crystalloids between the fluids which they separate, but they prevent the interchange of molecules as large as those of the blood colloids. Since blood colloids cannot normally escape from the blood stream, they must exert their osmotic pressure to prevent water being expressed from the blood, and to draw surrounding water into the blood. Consequently, fluid exchange between blood and aqueous appears to be the resultant between the physico-chemical forces which regulate the semi-permeability of the capillary walls and of the ciliary epithelium, the osmotic pressure of the blood colloids, the blood pressure within the ocular capillaries, and the intraocular pressure. It is suggested that the capillaries of the ciliary body carry a blood pressure sufficiently high to overcome the intraocular pressure combined with the osmotic pressure of the blood colloids, thereby giving rise to the aqueous as an ultrafiltrate. It is suggested that the capillaries of the iris and the collateral vessels draining Schlemm's canal carry a blood pressure lower than this critical level, thereby effecting an osmotic reabsorption of aqueous into the blood stream here. With so many variables, it is possible that the formation and reabsorption of aqueous need not progress at a uniform rate.

Primary glaucoma may arise from an imbalance in these forces, whereby fluid inflow is increased, or fluid outflow is decreased, or both, probably taking place as follows:

1. *Increased inflow.* It is suggested that the ciliary epithelium in glaucoma becomes abnormally permeable to all the constituents of the blood. When inflow exceeds the total available outflow, there must be an increased intraocular pressure.
2. *Decreased outflow.* It is suggested that the blood colloids may pass through the ciliary epithelium in glaucoma. Ferments in the

[1] Read before the New England Ophthalmological Society, April 12, 1927.

aqueous probably split the colloids into molecules small enough to escape by diffusion into the blood stream. But the blood colloids which are caught within the colloidal framework of the vitreous are probably more shielded from ferment action; hence the vitreous is caused to swell, the anterior chamber is made shallow, and the root of the iris is brought in contact with the pectinate ligament. In this way the surface available for outflow of aqueous is decreased, and the intraocular pressure rises.

3. *Vicious cycle.* Since iris blood drains into the venæ vorticosæ, an intraocular pressure increased by either method must compress the uveal vessels against the unyielding sclera, thus raising the blood pressure within the iris capillaries and establishing a higher level at which intraocular fluid exchange can take place.

I. *Fluid exchange in living organisms.*

An animal can survive the loss of nearly all its fat, of nearly all its carbohydrate, of half its protein, but of only one-fifth its water content. Water is indispensable for living organisms because it provides an environment so suitable for vital functions. Next to mercury, water has the highest surface tension known. Water is a universal solvent, dissolving more solids, liquids, and gases than any other known solvent. It takes more heat to raise the temperature of a gram of water one degree than is required to raise the temperature of a gram of any other substance one degree, excepting only liquified ammonia. On account of its high specific heat, water acts as a temperature stabilizer for the living organism, which cannot endure wide and quick temperature variations. Water is an excellent ionizing medium, and it thus accelerates all kinds of chemical reactions between its solutes. Water is not only the physical basis of protoplasm, but it provides the vehicle for the transport of oxygen, foodstuffs, and waste products to and fro within the living organism.

Exchange of water and other substances between blood and tissue fluids takes place through the walls of the capillaries. These exchanges are mediated by the hydrodynamic pressures within and without the capillaries, by the osmotic pressure of the fluids inside and outside the capillary tubes, and by the physico-chemical forces which bring about the semi-permeability of the capillary walls.

Krogh (1) and his workers have "rediscovered" the capillaries in the sense that capillaries can no longer be re-

garded as passive tubes, because their walls possess an independent contractility. Krogh states that "blood cannot be forced from arteriole into capillary until the walls of the capillary first relax." In the low pressure capillaries of the skin, Lewis (2) has shown that entrance of blood can be resisted with a force equal to 60 millimeters of mercury, and that expulsion of blood can be accomplished with a force equal to 90 millimeters of mercury. Vimtrup (3) has proven that contractility of capillaries is a function of the muscle cells (Rouget cells) applied here and there around the endothelial tube, and supplied with a net of non-medullated nerve fibers connected with the sympathetic system. Krogh and Rehberg have demonstrated that capillaries dilate in very weak concentrations of carbon dioxide, and that capillary tonus is maintained by pituitrin in dilution of one part per million, a dilution which has no effect whatever on arterioles. Hence it would appear that capillaries play a purposive, and a hitherto little appreciated part in the vital exchanges which are constantly taking place between blood and tissue fluids.

Of all complex factors involved in exchanges between blood and tissue fluids, the most decisive factor is undoubtedly the semi-permeability of the capillary wall. Semi-permeability is still one of the great puzzles, which the colloidal chemists are investigating. Perhaps the simplest conception of permeability is that of porosity, in which the size of the pore and the electrical charge it bears determine what can pass through. Of course the permeability of a living membrane is not so simple as this, and indeed it has no fixed value, but it varies with alterations in surface tension, absorption, hydrogen ion concentration, temperature, and pressure of the adjacent fluids. In general, it may be accepted that capillary walls and animal membranes are semi-permeable, passing easily the small molecules of water and crystalloids, and holding back the much larger molecules of the proteins (mol. wt. 30,000). The impermeability of animal membranes to protein is only relative and not absolute; pericardial fluid may contain up to 28 parts of protein per thousand and show spontaneous clotting, pleural fluid up to 60 p. M. with spontaneous clotting, while aqueous and cerebrospinal fluid normally contain only a very slight trace of protein and rarely clot.

It would seem that the membranes of the eye and brain are especially impermeable to protein except when diseased or injured, and that here this impermeability might be a biological adaptation designed to promote a normal fluid exchange.

Potential osmotic pressure cannot become evident or effective until the osmotic substance is placed within a membrane which is impermeable to it. Within a membrane of copper ferrocyanide, impermeable to crystalloids, the crystalloids of the blood exhibit an osmotic pressure equal to nearly 5000 millimeters of mercury. Within an animal membrane, permeable to crystalloids, the crystalloids of the blood exhibit no osmotic pressure whatever, or at least only a very transient one. This is because the molecules of crystalloid by diffusion rapidly escape from their confinement within the membrane, and they soon reach an equilibrium on either side of the membrane.

Since capillary walls in health are almost impermeable to blood colloids, it follows that blood colloids can exert their full osmotic pressure in the process of fluid exchange between blood and tissue. Starling (4) first called attention to this fact, and he first ascertained that blood colloids possessed an osmotic pressure equal to 30 millimeters of mercury. More perfect recent measurements have yielded nothing but verification of this value. Reid (5) has claimed that the osmotic pressure of the blood colloids is not due to the colloids themselves, but to the crystalloid adsorbed upon the colloidal surface, because repeated dialysis in distilled water removes the crystalloid and reduces the osmotic pressure of the purified colloid almost to zero. However, it seems that one is dealing here with the isoelectric point of the colloid, where surface tension and osmotic pressure reach their minimum.

From the foregoing paragraphs, and for simplicity if one assumes that the osmotic and hydrodynamic pressures outside the capillary are equal to zero, then it will become self-evident that exchange of fluid between capillary blood and tissue fluid will be the resultant between two constantly opposing forces within the capillary; on the one hand that of hydrodynamic pressure, and on the other hand that of osmotic pressure of the blood colloids. Bayliss (6) has applied this concept to lymph production, hypothesizing for each capillary

a "secreting" end where fluid is expressed from the blood because hydrodynamic pressure exceeds osmotic pressure, and an "absorbing" end where tissue fluid is osmotically sucked up into the blood because osmotic pressure exceeds here the hydrodynamic pressure. The decisive factor is of course the osmotic pressure of the blood colloids, to which the capillary wall is impermeable.

A very instructive capillary anatomy and physiology has been worked out for the kidney, and it seems to support the theory of fluid exchange stated above. Two sets of capillaries, a high pressure set, and a low pressure set, are strung along the course of the renal artery. The afferent branches of the renal artery are fairly large and they carry a high pressure. After breaking up into the glomerular capillary tuft, there is a single continuation in the efferent vessel, which is only two-thirds as large as the afferent branch. This efferent vessel shortly divides into capillaries which surround the tubules, and thence blood is drained off by way of the renal veins. It has been estimated that 61 liters each day pass through the capsule of the glomerulus from the blood, and that 60 liters of this ultrafiltrate are reabsorbed from the tubules into the blood each day. The glomerular capillaries must carry a high pressure, because of the size and contained pressure of the afferent artery, and because of the reduced size and the increased viscosity of the blood of the efferent artery. For just the opposite reasons, the capillaries of the tubules must carry a much lower pressure. If the general blood pressure is reduced to 40 millimeters of mercury, or less, there is a total suppression of urine. If the blood pressure is maintained at this low level, and if the blood is diluted by injecting normal saline, the flow of urine is reinstituted, probably because there is a new level of osmotic pressure of the blood colloids to be overcome. If the ureter is ligated, urine will continue to be formed until the intra-ureteral pressure rises to 90 millimeters of mercury, with the blood pressure at 130 millimeters, when urine formation will cease. Here seems to be a striking example of the decisive part which the osmotic pressure of the blood colloids plays in fluid exchange.

(*To be concluded.*)

TWO CURIOUS OPTICAL ILLUSIONS.

By Dr. G. LINDSAY JOHNSON, Durban, So. Africa.

(With three illustrations in the text.)

I DISCOVERED the two following illusions by accident, and as they have as far as I know, never been mentioned before I think they may prove of interest.

The first illusion may be seen by anybody in almost any restaurant and forms a proof of Listing's Law. As some of our readers may not be familiar with this Law, I will briefly describe it. If we sit down in front of a white wall at a little distance, and first fix a bright red vertical cross in front on the wall in such a position that when our line of sight is at right angles to the wall, it shall be directly in front of us. Then the visual field will be determined for the direction of fixation. This is far from being evident a priori, since it might be supposed that the eyeball is capable of rotation round its visual axis. But this never occurs beyond a small angle not exceeding at the most two or three degrees. So long as the head is rigidly kept in a vertical position, the eyes may roll in any direction, but the cross will always assume a vertical position, and not twist in any direction. But if we turn the head away from the primary position upwards or downwards or sideways to what we may call a secondary position, and then keeping the head in that position we gaze steadily at the cross for some moments (so as to obtain an after-image in the complementary color), and then turn the eyes directly upwards and keep them still, we shall find that the cross remains upright as before, but directly we turn the eyes sideways to the right, the cross appears to turn partly round the vertical arm turning through about 30 degrees to the right. So there is only one position

of the head in which the cross remains erect; for every other position the cross turns partly round. This is the Law of Listing.

Now if we look at a ventilator fan having two or four blades, such as can be found in almost any restaurant, which must be at a considerably higher level than the eye, and we sit about fifteen or twenty feet from it, and then raise our eyes so as to look vaguely (*i.e.*, without focussing the eyes) in the direction of any spot about two or three yards *above* and to the *right of* the ventilator, the fans which were revolving clockwise, will appear to revolve *in the opposite direction* and to be tilted downwards and outwards at an angle of approximately 30 degrees from the horizontal plane.

I have tried this experiment hundreds of times, as have a number of my friends, and we have invariably found this to be the case. I think this can readily be explained by Listing's Law, but the reason why the fans appear to revolve in the *opposite* direction, is due to another cause, and the solution is I think due to the fact that the center of rotation is not the same as the nodal point, in the optical system of the eye, but is situated 5*mm* behind the nodal point or points (for they are close together), whereby movements of objects out of the primary line of regard appear to turn in the opposite direction, when the head is not placed in the primary position. But this may not be the true explanation, at any rate it is not quite as satisfactory as is that of the tilting of the fans. But although not entirely satisfactory, I fail to discover any other rational explanation of the phenomenon.

The second optical illusion is as follows:—

If you look at any isolated raised spot about the size of a pin's head or somewhat larger, (and such spots formed by tiny blobs of whitewash or paint are very numerous), on a white wall about a foot or fifteen inches in front of you, and then raise the eyes slowly, and look vaguely into space without focussing the eyes, the spot will appear to travel in an irregular fashion upwards, exactly as an insect would do. In fact the illusion is sometimes so perfect as to deceive one into thinking that it is really an insect. But the moment one fixes one's eyes on to the spot the illusion disappears and the spot ceases to move, and its real nature is seen at once. I think that this

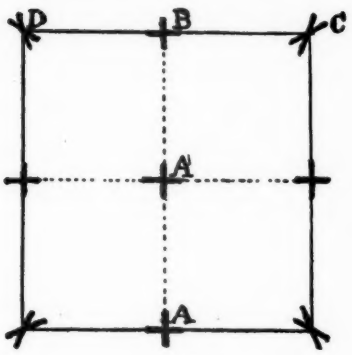

Fig 1.

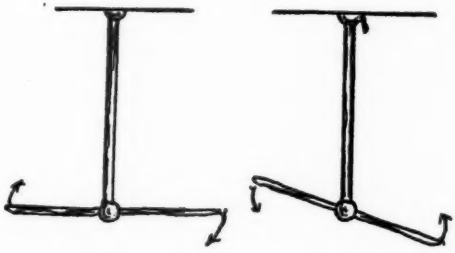

Fig 2. Fig 3.

EXPLANATION OF FIGURES.

Fig. 1. A. Primary position of the eye.
Axis of vision at right angles to the wall.
B. Secondary position of the eye. Head raised so that the axis of vision is no longer at right angles to the eye.
C. and D showing the apparent rotation of the cross through approximately 30 degrees.

Fig. 2. Real position of the ventilator fans.

Fig. 3. Apparent position of the fans when the eye is turned upwards and outwards from the fans. The image is not seen in this case by direct vision but by a part of the retina at some little distance from the macula, and therefore somewhat indistinctly, but well enough to make out the direction of rotation and inclination of the fans.

apparent movement is likewise due to the fact that the eye after it has been raised will slowly but unconsciously droop, thus causing the back of the eye and with it the macula to rise, and so the spot appears to run up the wall in an irregular manner. This very important fact that the eye does move in tiny jerks, and not in a steady sweep, was first discovered by Dr. Mulder, an ophthalmic surgeon practising in Durban, Natal, and repeated experiments by myself have confirmed his observation in the fullest manner.

CAUSES OF BLINDNESS IN MISSOURI AND IN ST. LOUIS.

By Dr. H. D. LAMB, St. Louis, Mo.

IT will be remembered that the granting of a pension of $25.00 a month to the adult blind in Missouri was begun in 1921. The Fifty-first General Assembly of Missouri in 1921 established the visual limit at $\frac{20}{200}$, that is no one with vision better than $\frac{20}{200}$ was eligible to receive the pension. Within this limit there were examined and admitted to the pension-rolls about 6,000 adult blind. This number was about twice as many as could be paid from the tax-money available. It was found that about one-half of these applicants had vision no greater than light perception. Accordingly in 1923 the Fifty-second General Assembly changed the limit of vision to light perception. Every blind pensioner was RE-EXAMINED BY AN EYE-SPECIALIST ONLY. The following tables are therefore the findings in summary among blind adult applicants for the pension, BASED ENTIRELY ON NEW EXAMINATIONS begun about June 1, 1923. About two-thirds the number listed in the following tables are eligible and receive the pension. For statistical purposes, there is no reason to exclude the findings among ineligible applicants. We are interested in obtaining as many examinations of blind individuals as possible made by specialists.

Because a single eye is blinded in many cases from a different cause from what has cost the sight of the fellow-eye, it becomes necessary to list cases under each cause of blindness as bilateral and unilateral cases. (Tables No. 1 and No. 4.)

For the ages of the blind in Missouri, I have listed 5392 persons out of the total 5552 individuals; the ages of the remaining 160 were not easily available.

TABLE NO. I.—CAUSES OF BLINDNESS IN MISSOURI.

Causes	*Persons blinded in both eyes* (*No.*)	*Single eyes blinded* (*No.*)	*Eyes in total* (*No.*)	*Cause of blindness* (*Per cent*)
Trachoma	1380	76	2836	25.5%
Cataract	915	275	2105	19.0%
Optic atrophy	589	95	1273	11.5%
Glaucoma	421	84	926	8.3%
Trauma	175	335	685	6.1%
Corneal ulceration	200	197	597	5.3%
Uveitis	210	159	579	5.2%
Choroiditis and chorioretinitis	163	44	370	3.3%
Ophthalmia neonatorum	99	5	203	1.8%
Retinal degeneration	48	5	101	0.9%
Myopia	38	16	91	0.8%
Optic neuritis	29	5	63	0.6%
Retinal detachment	15	19	49	0.4%
Congenital cataract	18	3	39	0.4%
Hydrophthalmus	9	2	20	0.2%
Microphthalmus	6	1	13	0.1%
Amblyopia ex anopsia		8	8	0.1%
Keratoconus	2		4	0.05%
Miscellaneous	3		6	0.05%
Fundus condition (obscured by lens changes)	61	38	129	1.5%
No diagnosis	323	329	975	8.9%
Totals	4704	1696	11,104	100.00%

Total number of persons..... 5552

NUMBER OF BLIND ADULTS IN MISSOURI WITH VISION OF 0 AND LIGHT PERCEPTION..... 3630 65.4%

TABLE NO. II.—AGES OF BLIND IN MISSOURI.

Ages	*Number of cases*	*Percentage of cases*
From 20 to 30 years	219	4.1%
From 30 to 40 years	411	7.6%
From 40 to 50 years	610	11.3%
From 50 to 60 years	818	15.2%
From 60 to 70 years	1248	23.1%
From 70 to 80 years	1318	24.4%
From 80 to 90 years	636	11.8%
From 90 to 100 years	122	2.3%
From 100 & over	10	0.2%
Totals	5392	100.0%

TABLE No. III.—SEX OF BLIND IN MISSOURI.

Sex	*Number of cases*	*Percentage of cases*
Males	2979	53.7%
Females	2573	46.3%
Totals	5552	100.0%

TABLE No. IV.—CAUSES OF BLINDNESS IN ST. LOUIS.

Causes	*Persons blinded in both eyes* (*No.*)	*Single eyes blinded* (*No.*)	*Eyes in total* (*No.*)	*Cause of blindness* (*Per cent*)
Optic atrophy	147	1	295	23.1%
Glaucoma	99	4	202	15.8%
Uveitis	61	8	130	10.1
Corneal ulceration	55	7	117	9.1
Cataract	49	7	105	8.2
Simple trauma	24	8	56	4.4
Trauma with sympathetic ophthalmia	12		24	1.9
Pigmentary degenerations of retina	35	1	71	5.6
Ophthalmia neonatorum	29		58	4.6
Trachoma	22	3	47	3.7
Chorioretinitis	14	3	31	2.4
Congenital malformations	14	1	29	2.3
Progressive myopia	12	4	28	2.2
Retinal detachment	5	4	14	1.1
Congenital cataract	6		12	1.0
Acid, lime and lye burns	6		12	1.0
Essential shrinking of conjunctiva	4		8	0.6
Parenchymatous keratitis	3		6	0.5
Optic neuritis	2		4	0.3
Amblyopia ex anopsia		4	4	0.3
Wood alcohol	1		2	0.2
Miscellaneous causes	4	2	10	0.8
Fundus condition (obscured by lens changes)	3	1	7	0.5
No diagnosis		4	4	0.3
Totals	607	62	1276	100.0%

638 Individuals in all

We have never published any separate statistics on blindness for the city of St. Louis. It is interesting however to compare the causes of blindness for this large city with those of the entire state.

TABLE No. V.—VISION OF BLIND IN ST. LOUIS.

Vision	*Number of cases*	*Percentage of cases*
0 and light perception	465	72.9%
Light perception to 10/450	131	20.5
10/450 to 20/450	42	6.6
Totals	638	100.0%

TABLE No. VI.—AGES OF BLIND IN ST. LOUIS.

Age	*Number of cases*	*Percentage of cases*
From 20 to 30 years	48	7.5%
From 30 to 40 years	72	11.3
From 40 to 50 years	100	15.7
From 50 to 60 years	127	19.9
From 60 to 70 years	138	21.5
From 70 to 80 years	108	17.0
From 80 to 90 years	36	5.7
From 90 to 100 years	9	1.4
Totals	638	100.0%

TABLE No. VII.—SEX OF BLIND IN ST. LOUIS.

Sex	*Number of cases*	*Percentage of cases*
Male	345	54.1%
Females	293	45.9
Totals	638	100.0%

In brief, the striking difference between the causes of blindness for the entire state as compared with those for St. Louis is

in the amount of trachoma. The greatest single cause of blindness (25.5%) in the entire state, trachoma as a cause of blindness in St. Louis ranks ninth (3.7%). Trachoma is therefore a rural disease because of inadequate ocular prophylaxis and treatment in the country districts.

One would expect optic atrophy to be much more prevalent in St. Louis (23.1%) than over the entire state (11.5%) because syphilis, the predominant cause of optic atrophy is more common in the big cities than in the rural places.

That there is more blindness from glaucoma in St. Louis (15.8%) than in the entire state (8.3%) is surprising and hard to believe. It can only mean that there is more glaucoma in St. Louis than generally over the state.

The reasons for drawing up and publishing another set of statistics for Missouri are first that the above figures are based throughout on fresh or new examinations of each applicant's eyes and secondly, that these new examinations were all made by eye specialists. These figures correspond well with the former statistics of blindness in Missouri published in the Journal of Missouri State Medical Association for May, 1926.

BILATERAL PREPAPILLARY VASCULAR LOOP OF THE RETINAL ARTERY.[1]

By Dr. ARTHUR M. YUDKIN, New Haven, Conn.

(With three illustrations on Text-Plate XVII.)

J. S., female, colored, age 14, was seen at the clinic for a refractive error; the vision in right eye $\frac{11}{8}$, left eye $\frac{11}{8}$. Under a mydriatic, a slight degree of hypermetropic astigmatism was found. With correction the vision became normal. The visual fields revealed no unusual scotomata. The media were clear and the fundi oculi appeared normal except for the following vascular abnormalities.

In the left eye the central artery of the retina divided on the disc into two main branches. From the lower one a spiral loop protruded into the vitreous for about 3–4 diopters. The outgoing vessel made a sharp loop and returned to upper temporal edge of disc after marking three distinct spiral coils about the retina in the normal position of the superior temporal artery. The recurrent part of the process was of a slightly darker hue than the retinal arteries. The whole loop pulsated synchronously with the radial artery. It did not change its position with the movement of the eyeball. The protruding anomaly was enclosed in a transparent whitish covering, quite noticeable at the apex of the process. The remainder of the blood vessels were distributed in the usual manner.

The right eye presented a similar vascular anomaly. The loop, however, was less marked and did not extend into the vitreous. A slit-lamp examination revealed no remnants of the hyaloid arterial system.

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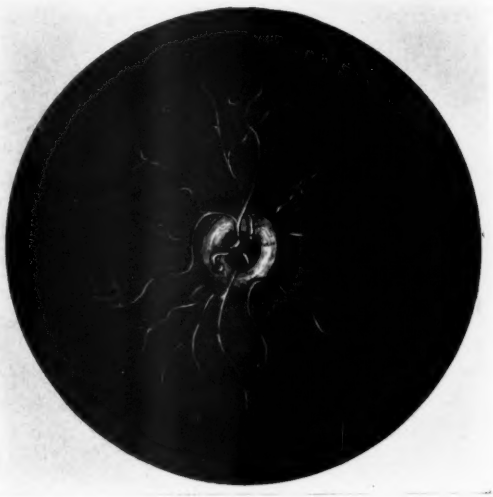
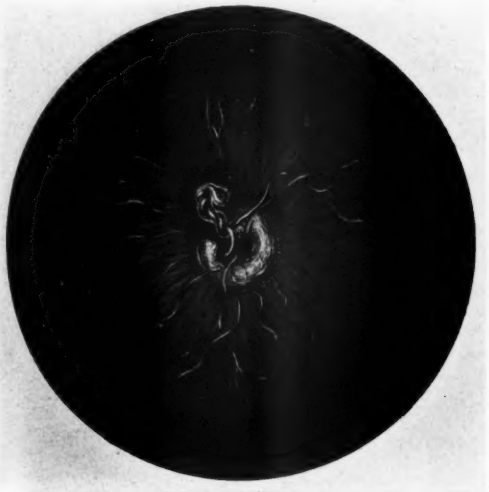
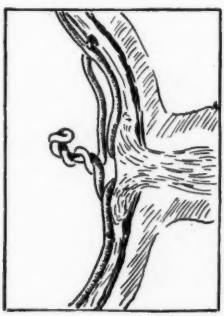
[1] Presented before Eye Section, N. Y. Acad. Med., March, 1927. From the Section of Ophthalmology, Yale School of Medicine.

ILLUSTRATING DR. ARTHUR M. YUDKIN'S ARTICLE "BILATERAL PREPAPILLARY VASCULAR LOOP OF THE RETINAL ARTERY."



Prepapillary Vascular Loop of the Central Artery of Retina, (left eye).





Prepapillary Vascular Loop of the Central Artery of Retina (right eye).

Comment.

Similar monocular vascular anomalies have been reported in the literature, but bilateral conditions of this type are rather rare. Most of these cases were described as examples of persistent hyaloid arteries or veins. Liebreich (1) thought his case of vascular loop in the vitreous originated as a hyaloid artery and terminated as a hyaloid vein on the disc. It has been definitely shown by Von Hippel (2), that the hyaloid artery did not take such a course and a vena hyaloidia was unknown. He was supported by Leber (3) in his contention that these vessels were not remains of an embryonic circulation.

Merrill and Wagener (4) found several cases of an anomalous spiral loop of the retinal vein in the vitreous described in the literature and added a case of their own. Many other reports of aberrant circulation in the vitreous have also been published but they were definitely due to inflammatory processes. Hirshberg (5) was the first to describe this vascular loop as a retinal artery. His conclusion has been corroborated many times with case reports.

Kipp and Von Hippel (6) suggested that the spiral loops were derived from the retinal vascular system and that the abnormality was an exaggerated tortuosity of these vessels.

The tissue covering the loop has been described as a remnant of the hyaloid system and it has been considered as the internal limitating membrane of the retina, with perhaps a portion of the nerve fiber layer pushed ahead or carried along with the retinal vessel in its process of outgrowth into the vitreous.

I am in accord with the opinion presented by Kipp and others, that this vascular anomaly is probably an excessive development and an exaggerated tortuosity of the central arterial system, and is not due to an inflammatory condition nor is it the remains of the embryonal hyaloid artery.

The tissue covering, is probably the outer coat of the vessel reinforced by the hyaloid membrane which the structure has pushed ahead of it.

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VASCULAR LESIONS OF THE FUNDUS OCULI.[1]

By Dr. W. T. DAVIS, Washington, D. C.

IN arteriosclerosis the lesions may be observed so early in the fundus of the eye that the ophthalmologist is often the first to suggest a diagnosis. Likewise, he is frequently the first to be consulted by patients with this disease, due to the early incident of ocular symptoms. One of these early ocular symptoms is persistent and obstinate asthenopia. This occurs frequently before general symptoms of vascular disease have appeared. While the refraction is being done there is alternate dimming and clearing of the test objects. This may occur while a change of the test lenses is being made, rendering it exceedingly difficult to make a subjective examination. After the most painstaking and careful refraction these patients return still uncomfortable. No matter what glass is given the profound asthenopia continues. These cases differ from nervous asthenopia in which somewhat like symptoms are present. In the arteriosclerotic reaction the nervous element is absent both in history taking and during refraction. Nervous asthenopia is not real asthenopia but a symptom of a nervous derangement, while the arteriosclerotic condition is real and persistent asthenopia brought about by arterial disease. The cause of this symptom is, no doubt, innutrition of the retina, hence the dysfunction. The vascular changes in arteriosclerosis gradually obliterate the capillaries and arterioles and lessen the caliber of the larger vessels. This greatly lessens the blood supply of both retina and chorioid.

According to Salzman the capillary network of the retina

[1] Read before Joint Meeting of Ophthal. Socs. of Baltimore, Md., and Washington, D. C., at Baltimore, Jan. 27, 1927.

consists of very narrow capillaries; fine branches are given off at right angles which break up into capillaries 13 to 25*mm* from the main vessels. In this way a space free from capillaries arises on each side of the main vessel. The capillaries go no farther than the outer surface of the inner nuclear layer, hence the border between the first neuron (rods and cones and the outer nuclear layer belonging to them) and the second neuron (the nerve cells of the inner nuclear layer) is also the dividing line between the avascular portion of the retina, the sensory epithelium, and the vascular portion, the cerebral layers of Schwalbe. Along with the cerebral layers the retinal vessels fail in the center of the fovea where a capillary free area of about 5*mm* of irregular shape is formed.

The larger vessels of the chorioid do not form a rich anastomosis though the ophthalmoscopic appearance would seem to indicate they do. This appearance is brought about by the frequent crossings of the vessels. Since we see the vessels indistinctly, and their walls not at all, we look upon these crossings as anastomoses. In the vessel layer of the chorioid the caliber of the vessels decreases from without inward. In the capillary layer (chorio-capillaris) the capillaries lie in one plane, *i.e.*, they do not form a meshwork, but a net which is characterized by the width of the individual vessels. This network is especially thick in the foveal region.

The endarteritic process gradually obliterates the capillaries and small vessels both of the retinal and chorioidal systems. This endarteritis may result, according to Parsons, from localized interference with the blood flow. It may also be caused by septic and toxic processes. It is conceivable that interference with the chorioidal circulation might cause such changes in this structure, thus interfering with the function of the outer layers of the retina, while the retinal vessels remain normal. The interference might for example occur from the increased intraocular tension in glaucoma, preventing free exit of blood by way of the venæ vorticosæ. This innutrition of the outer part of the retina may account in part for some of the subjective symptoms in glaucoma.

Fuchs says degeneration and sclerosis of the vessels in high myopia take place from the stretching; thickening and obliteration of the vessels occur. Parsons says primary arterio-

sclerosis is not uncommon in the retina. Hence it is a question of changes in the very small vessels. It is characterized by proliferation of the endothelium of the intima and new formed connective tissue, especially elastic fibers. This is the essential histologic feature. The increase of elastic tissue continues throughout life, but it is the irregularity of it which is indicative of disease. The endothelial proliferation may be uniform so that the lumen of the vessel may be concentrically diminished. More commonly it is irregular so that knob-like excrescences project into the lumen which is finally obliterated. The swelling consists at first of cells chiefly derived from the epithelium, partly from connective tissue. There is a superficial covering of epithelium which is lost at a later stage. There is colossal increase in the elastic tissue fibers in these knobs; they also extend into the media, the muscle cells of which seem to divide before them. The longer the process lasts the denser becomes the perivascular connective tissue. Marked vascular changes are not inconsistent with normal vision and absence of ophthalmological signs. In the advanced cases with silver wire arteries the endovascular changes are more marked in the arteries, the perivascular changes in the veins. The latter rarely show excrescences into the lumen, but show an infiltration of the adventitia with round cells. The infiltration of the walls of the arteries is rarely pronounced.

In retinitis pigmentosa the obliterative changes are marked; these are principally in the arterioles, the walls of which are thickened and deeply pigmented, the lumen finally becoming blocked. Œdema of the retina is seen with the ophthalmoscope as a grayish veil covering the fundus, more especially in the neighborhood of the disk, along the course of the vessels or in scattered areas over the fundus. This veiling of the retina is very characteristic and is frequently the first noted pathological detail.

Foster Moore regards the primary change in arteriosclerosis as beginning in the arterioles and smaller arteries, producing increased resistance to the onward passage of blood, causing innutrition of the tissues. If the ocular vessels are the subjects of sclerosis and the blood pressure within them be not compensatorily raised, the retinal functions are likely to be

impaired. Knapp summarized the common beliefs regarding the prognostic significance of sclerotic changes in the retinal vessels. In this summary are the following important points; angiosclerosis of the retinal vessels indicates the same condition of the cerebral vessels; slight changes in the retinal vessels are important if associated with other symptoms of arteriosclerosis; in diabetes and nephritis there are changes in the retina due to angiosclerosis which are independent of and give a different prognosis from the albuminuric and diabetic retinitis. Indeed it would appear that the prognosis is bad in diabetic retinitis only after vascular disease has made its appearance.

In those with retinal arteriosclerosis temporary obscurations of visions are gravely important. In Bright's disease the vascular changes are of decided and early importance as regards the diagnosis and prognosis. It is agreed by most that Bright's disease is primarily one of the vascular system. The vessels of the kidney may be early involved leading to the parenchymatous and interstitial changes due to innutrition. Instead of the early involvement of the kidney the disease may manifest itself in the vessels of the brain or elsewhere, and in the case of the last named, the retinal vessels may be among the first involved.

Hypertension of the arteries is thought to be of toxic origin and when long continued may cause changes in the vascular system known under the term arteriosclerosis. Toussig thinks, however, there is a toxin directly increasing the arterial tension and at the same time having an effect on the vessel wall. Where the action of this toxin is of long standing arterio-capillary sclerosis will occur. It is thought, however, by Gull and Stratton that Bright's disease, particularly the interstitial form, is a manifestation of general arteriosclerosis in which the kidney symptoms are the predominating ones. No advanced arteriosclerotic changes exist without involvement of the kidney to some extent. Whether the renal lesion will be the predominant factor depends on the power of the kidney to resist the effect of the toxin which is producing the vascular alteration. It is thus apparent that involvement of the retinal arterial system by this sclerotic change indicates the process is in motion within the body. Hence it assumes

paramount importance in the diagnosis of the condition with the ophthalmoscope, as the treatment that is of the greatest avail is that which is instituted early.

Weeks, discussing albuminuric retinitis divides the condition into two classes; first, those not preceded by thickening of the vessel walls, which will include cases occurring as a result of acute parenchymatous nephritis in which the changes in the kidneys precede those in the retina. Second, those cases in which hemorrhages and exudation are preceded by changes in the walls of the vessels. Very early retinal lesions due to toxic influences are as follows—small, almost indistinguishable white dots about the disk in the neighborhood of the macular; haziness and delicate hyperemia of the disk; isolated, faintly milky areas in the retina and beaded appearance of some of the larger vessels with or without visible alterations; one or all of these lesions may exist without perceptible alterations of the vision.

Spasm of the retinal vessels is probably an arteriosclerotic manifestation; vision may be lost for a few minutes to some hours, the appearance of the retina resembling that seen in occlusion of the retinal artery, due to the pronounced ischemia. Should this ischemia last sufficiently long the result will be the same as in occlusion; in any event, spasm of the vessels is indicative of serious arterial disease and is a bad prognostic sign. A certain amount of damage is done to the delicate structure of the retina, even though the vision is not seriously impaired by the attacks. It would seem probable that some cases reported as dislodged embolus might be spasm.

If the central artery is thickened by sclerosis the vein becomes displaced and compressed. Occlusion of the vein results from proliferation of the endothelium and sub-endothelial tissue; this may give a typical picture of venous thrombosis. Some important hemodynamic effects take place as a result of the close amalgamation of the two vessels.

The pulsation of the artery against the vein may cause acceleration of the circulation in the vein and may account for the pulsation of the veins on the disk.

Charlin and Munoz consider that vascular degeneration and disease cause glaucoma. These changes begin first in the periphery of the retina and the cupping of the disk occurs

under normal ocular tension as the result of these changes. All the fine vessels of the retina reveal thickening of the walls with hyaline degeneration and loss of the lumen. These findings are confirmed by Schnabel.

Rönne describes a form of arteriosclerotic atrophy due to retinal changes and differing from the ordinary form in the changes in the visual fields. There are illy defined defects in the visual field. In the beginning there are central or paracentral scotoma so that the condition may be mistaken for retrobulbar neuritis. Both eyes are usually affected after an interval. The ophthalmoscope shows changes of the caliber of the arteries; the blood column is irregular with varying diameters and at certain points threadlike, but does not entirely break off. The sudden impairment of central vision is thought to be due to small and incomplete infarcts in the vicinity of the fovea produced by occlusion of the branches of the macular artery by the sclerotic process. In atrophy of the optic nerve due to arteriosclerotic spasm, widening of the visual fields may be brought about by giving atropin subcutaneously or by mouth. This will not occur in other forms of atrophy.

Chabonier thinks there is no casual relationship between retinitis and kidney insufficiency, they are rather parallel phenomena. Bright's disease is not purely a kidney affection; the principal changes are in the blood plasma, hypercholesterolemia is one of the main features. Wagner says patients with retinitis associated with arterial hypertension and moderate peripheral arteriosclerosis with little or no renal insufficiency form a distinct group, differing from those with chronic nephritis. The constriction of the retinal arteries is part of a general vascular spasm which is the cause of the high blood pressure; albuminuric retinitis must therefore be considered as angiospastic retinitis. From the general standpoint there is no doubt as to the association of retinitis with arterial hypertension, arteriosclerosis and Bright's disease. It would appear that such retinitis might be due in certain cases to innutrition of the retina, brought about by the obliteration of the capillaries and arterioles by the arteriosclerotic process. This reduction of the blood supply, in addition to the innutrition, must have important effects

on the delicate cellular elements of the retina and chorioid due to the reduced oxygen supply. There must be other causes in which retinitis is the result of the high toxicity of the blood acting directly upon the terminal vessels and retinal tissues; this would seem to be true in certain cases of acute retinitis as seen for example in retinitis gravidarum. There is still another group where the two factors may act in unison. The hemorrhages and exudates resulting from the vascular disease, while the actual inflammation of the retina may come from the toxic content of the blood. There are undoubtedly cases where these primary changes occur first in the retina or at least in this part of the cerebral circulation.

It is the opinion of Parsons that retinitis proliferans is due to hemorrhage into the retina, or preretinal hemorrhage plus actual retinal inflammation. Unless this combination be present proliferating retinitis will not occur. This condition is most likely to occur on and in the vicinity of the disk where the mesoblastic tissue is most abundant; it is only from the mesoblastic tissue that the proliferating process can start. There is abundant evidence to show that the greater part of the new-formed fibrous tissue in inflamed or degenerated retinæ is of mesoblastic origin derived from this tissue on the disk and from the walls of the blood vessels. The nearer the hemorrhage to the disk and the more widespread its traumatic and irritating effects in this immediate neighborhood, the more will proliferative action be excited and the greater will be the proliferation and organization. Hence the fact that retinitis proliferans invaribly springs from the disc or its vicinity. In the later stages of syphilitic retinitis the changes are mostly in the walls of the vessels, sometimes so great as to close the lumen. These changes are not pathognomonic.

It is questionable if the changes in diabetic retinitis are due to other than vascular injury. Diabetic retinitis may occur when the patient is in apparent good health. In several instances this condition has been observed as the first manifestation of the disease, and was the cause of the individual seeking medical advice. What is termed diabetic retinitis is not always an inflammatory disturbance in this organ but has its inception apparently in the vascular changes, the actual inflammation occurring later. One sees in what is otherwise

an apparently normal retina innumerable pin point, perfectly round hemorrhages scattered throughout the fundus. These hemorrhages are so small that they may be easily overlooked.

Jervey mentions a form of retinitis which he thinks is of vascular pathogenesis. There is non-hemorrhagic hyperplastic exudation which is not due to syphilis, tuberculosis or trauma. The gouty retinitis appears to be entirely due to the arteriosclerotic changes induced by gout. Leber's pseudonephritic retinitis occurs sometimes in gouty subjects. It presents the star shaped figure at the macular and closely resembles albuminuric retinitis but is not due to renal disease. Periphlebitis retinalis is probably of tubercular origin in the majority of cases. The condition may involve the retinal veins extensively or only the small veins in the periphery. Retinitis proliferans may be present also. The condition would appear to be one of toxic origin in which there are vascular changes associated with actual inflammation of the retina. The periphlebitis would indicate the former, the retinitis proliferans the latter.

In a diseased organism according to Hanke, the tubercle bacillus prefers that organic system first attacked. He refers to the essential vascular tuberculosis of Banda. To this condition belongs the retinal periphlebitis of adolescence described and regarded as tuberculous by Axenfeld and Fleisher, and to this also belong the recurring hemorrhages of the vitreous body. That is, the vascular condition and the recurrent hemorrhages are correlative.

Thrombosis of the central vein of the retina or one of its branches is a not infrequent cause of profuse retinal hemorrhages, either throughout the retina or in the area supplied by the branch thrombosed. The thrombotic process may be brought about by pressure of the sclerosed central artery upon the vein near the lamina cribrosa where the vessels lie in juxtaposition. This pressure may cause thickening of the endothelium of the vein reducing the lumen and thus predisposing to the thrombotic process. Some intercurrent condition such as a fall in blood pressure or some infectious process as influenza for example, may precipitate an attack. Scattered hemorrhages throughout the retina when associated with swollen disc and retina and narrowed arteries make the

diagnosis thrombosis of the central vein justifiable, but do not show whether it is due to pressure from without or narrowing from within.

Embolism of the central artery will cause hemorrhages into the retina due to the acute anemia and consequent damage to the vessel walls. The closure of the artery is not always due to an embolus and this can be ruled out usually when on careful examination no cause for embolism can be discovered. The cause is often a thrombotic process in a diseased vessel. In endarteritis obliterans when the vessel is almost closed by the process a fall in the blood pressure will allow the walls of the vessel to make contact, thus shutting off the blood stream. Hence a careful analysis of the patient's general state of health is desirable if we are to obtain the best results in the treatment. Foster Moore and Gifford think thrombosis of the retinal vessels may follow ligation of the deeper vessels of the neck.

Hemorrhages into the retina will occur as a result of venous congestion from any cause; as a result of all forms of anemia the graver forms in particular, due to degeneration of the vessel walls; also the acute anemia induced by stoppage of the central artery. This stoppage may be due to spasm or hemorrhage into the optic nerve; rupture of the central vessel; inflammations in the orbit; facial erysipelas or influenza. Diseases inducing anemia are indirectly the cause of retinal hemorrhages or may be more directly the cause as a result of the toxemia. Conitano finds retinal hemorrhages frequent in malaria, even when quinin is used; he advises arsenic to offset the affect of quinin on the vessels and nerves.

Parsons quotes a case of persistent hyaloid artery which extended from the optic disk to the posterior surface of the lens; due to the elongation of the eye from myopia this vessel ruptured resulting in a profuse vitreous hemorrhage. Hemorrhage into the retina as a result of subarachnoid hemorrhage may be due to local affection of the arteries or a leakage of blood through the subpial lymph spaces around the lamina cribrosa and through the perivascular lymph spaces around the central retinal vessels. Chorioidal hemorrhages may result from sudden lowering of the tension of the globe due to operation on or to perforation of the globe, the vessels of which have undergone degeneration from disease; this is

particularly apt to occur in glaucoma. The expulsive hemorrhage following iridectomy for increased intraocular tension is sadly familiar. Chorioidal tumors may cause hemorrhage into the retina.

The most frequent cause of hemorrhage into the vitreous are injuries, chorioiditis, retinitis, myopia and diseases causing alterations in the blood such as diabetes and Bright's disease. Retinal hemorrhages in the newborn are probably more frequent than realized and may account for some of the amblyopic eyes seen later in life. These amblyopia cases in children will frequently show granular or finely pigmented maculæ. They will often gradually clear up and the vision reach normal or nearly so. While many of these cases are probably of toxic origin some are of hemorrhagic origin. Leukemia, hemophilia, scurvy, leprosy, botulism, relapsing fever, influenza, jaundice and the anemia induced by dibothriocephalus are among the causes or retinal hemorrhage; also poisons such as lead, phosphorus, CO_2, nitrobenzol, hydrastin, snake venom, etc.

In pernicious anemia hemorrhages are of some diagnostic value as they are uncommon in chlorosis; they also occur in the hemorrhagic diathesis and morbus maculosis, anomalies of menstruation, the menopause, uncinariasis and obesity. All forms of retinitis, particularly the syphilitic forms are associated with more or less hemorrhage into the retina. The neuroretinitis and choked disk resulting from increased intracranial pressure as a result of brain tumor or other causes is accompanied with marked retinal hemorrhage. Diseases of the chorioid, as disseminated chorioiditis and exudative chorioiditis are complicated with retinal hemorrhages. Ehuus mentions a large preretinal hemorrhage produced by two applications of radium. It is suggested that the X-ray may cause retinitis and consequent hemorrhage, but long exposure is necessary.

The disorganization of the vessel walls in severe retinitis gives rise not only to extravasations of plasma and leucocytes but also to hemorrhages varying from microscopic collections of red cells to extensive bleeding; small hemorrhages are first limited to the nerve fiber layer and are often arranged along the larger vessels. They do not necessarily arise from the

vessel near which they lie but the blood collects in or around the perivascular lymph sheaths, being drawn into this situation by the lymph flow. The nuclear layer is involved only when the hemorrhage is considerable. When the hyaloid is torn the blood involves the vitreous. When not extensive the blood spreads laterally, occupying the peripheral layers of the vitreous. Here it acts as foreign body, part being absorbed while the remainder remains encapsulated. The irritation of the clot causes the cells of the adventitia to proliferate. They grow over the clot forming first a thin membrane composed of a single layer endothelial cells; later the membrane is covered by a layer of young spindle shaped connective tissue cells which organize into dense fibrous tissue in the usual manner. Sometimes the blood shows little tendency to clot so that but little fibrin is formed. Absorption takes place by means of the phagocytic leucocytes which is a lengthy process. The hemoglobin is the most resistant; it slowly changes into a black pigment, which for a long time retains its iron constituent; it cannot then be distinguished from autochthonous pigment. The iron may be widely diffused especially in the elastic tissues of the vessel walls, but this is not as marked as in a true siderosis bulbi. The pigment is seen around the vessels particularly. In this manner retinitis pigmentosa may be accurately similated.

In young men, less often in young women, recurrent vitreous or retinal hemorrhages may occur over a long period of time, the commonest cause being tuberculosis. The other probable etiological factors are—gastrointestinal disturbances with constipation, syphilis, oxaluria, disturbances of the blood and menstrual disorders. Occasionally no cause can be found and these occupy a group to themselves; they are not tuberculous or syphilitic and appear perfectly healthy. On the other hand there are the tuberculous cases especially of the more chronic type or with bone and joint involvement where recurrent hemorrhages are common.

Aneurysms of the retinal arteries are rarely seen. That form most frequently seen are the miliary aneurysms in old people, as the result of arteriosclerosis and cardiorenal disease. Pringle mentions a case of aneurysms of the retinal vessels which was otherwise normal; the swellings appeared in the

extreme periphery of the field that could be seen with the ophthalmoscope. James divides miliary aneurysms into two groups; those due to senile changes in the vascular system. These show small aneurysms without inflammatory accompaniment and give rise to no changes in the retina except minute hemorrhages. In the other group the aneurysms are combined with gross changes in the retinal tissue and have a decided similarity to retinitis circinata. Usually the aneurysms are at the end of small vessels like berries on a stalk and may occur on the veins. The changes are always in that part of or near that part of the retina which is diseased. They are often surrounded by thin hemorrhages, rarely large. Leber points out that vision usually diminishes but is not lost unless secondary glaucoma sets in.

REPORT OF THE ANNUAL CONGRESS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

By Mr. HENRY DICKINSON, London.

The Annual Congress.

This year the Society held its congress in Liverpool, at the invitation of the Northern division of the Society, in the Medical Institution of that City. The chair was occupied throughout by the President, Sir John Herbert Parsons, F. R. S., and on the first morning the Lord Mayor of Liverpool attended and gave to the members a cordial welcome to the City, and gave a luncheon to them at the conclusion of the first morning session.

Papers.

Mr. E. Treacher Collins read a paper on **Metaplasia of the Tissues of the Eyeball.**

He said that metaplasia was the production of specialized tissues from cells normally producing tissues of other orders, owing to an alteration in environment, and not necessarily, nor even primarily, because of altered function. These changes seen in ocular tissues he divided into embryological, pathological, neoplastic and biological. An instance of the first of these was the formation of a new lens from the termination of the secondary optic vesicle in larval newts after destruction of the normal lens, and, in man, the substitution of skin for conjunctiva when the lids, in the course of their development, did not close over the eye. A further example was the substitution of retinal tissue for neuroglia in the optic nerve. In the pathological form occurred the formation of bone in

the choroid, and the substitution of fibrous tissue for the endothelium lining Descemet's membrane. He gave other instances. An example of neoplastic metaplasia was the formation of spindle-shaped cells in growths which were primarily endothelial; another was the mesoblastic tissue met with in connection with tumors of the lacrymal gland and of the orbit. The biological form included differences in the constitution of the sclerotic in various species of animals. For instance, the muscular tissue of the iris was composed, in some animals, of unstriated muscle fibres, in others of striated fibres.

Operation for Partially Contracted Socket.

This was the title of a paper by Mr. R. AFFLECK GREEVES. The essential feature of this procedure which the author had devised was the removal from the orbit of the fat and fibrous tissue, as if this was not done, the suturing did not seem to promise permanency.

MR. THOMSON HENDERSON (Nottingham) claimed to have devised and carried out a very similar operation ten years ago.

SIR ARNOLD LAWSON said he formed the idea, from Mr. Thomson Henderson's description of his own operation, that the result would probably be somewhat evanescent. Mr. Henderson, however, said he had had cases in which the result had lasted for years. The conjunctiva was pinned down to the skin, and it proved to be very permanent.

MR. G. MAXTED spoke of the difficulty caused by the silk cutting through the conjunctiva, especially if they had to bear tension.

MR. GRAY CLEGG described a modification which he had been employing, by passing a fine scalpel from the side and making a sagital section, so that without cutting in any way one could draw down the conjunctiva and form the sort of sulcus which Mr. Greeves pointed out.

The PRESIDENT said ophthalmologists would be grateful for a successful operation for even partially contracted socket. He referred to Weeks' operation for completely contracted socket, which he saw performed in America by its originator.

Mr. George Young read a paper on "Practical Keratometry."

Dr. P. Stibbe, lecturer on Anatomy, Liverpool University gave a lantern exposition on the

Comparative Anatomy of the Nictitating Membrane.

He said the purpose of this membrane was to protect and clean the cornea, as its function was to move rapidly to and fro across the front of the eye. In the bird the membrane was a crescentic structure covering the inner fourth of the margin of the cornea, and it was continued beneath the lower lid, and was connected with muscle at the back of the eyeball. Under the eyeball was a sheet of fibrous tissue slung like a hammock. Two small fibrous bands helped to keep the lower eyelid in position while the nictitating membrane moved. He discussed in considerable detail the mechanics underlying the movements of this nictitating membrane, and passed to a consideration of the function of the human plica semilunaris. He suggested it was an additional apparatus in possession of man, not for the purpose of protecting the cornea, but to protect the puncta lacrymalia and lacrymal canals, and to prevent foreign bodies which might enter the eye leaving that way. In the amphibian the lower lid consisted of a membrane part and a lid part. For the membrane in animals he preferred a name he had given it, "the intercepting membrane."

Mr. Treacher Collins discussed the lecture, and referred to the function of the protrusio bulbi. In serpents the third eyelid formed a permanent covering.

Discussion on Nystagmus (other than miners').

A discussion on this subject occupied the whole of Thursday afternoon, and was opened by Mr. B. Chavasse (Liverpool). Mr. Chavasse said the subject could be approached by various channels; it could be regarded as a movement, as a symptom, an adaptation, a habit, a neurological problem. True nystagmus, that evoked when the eyes were directed straight ahead, might increase in certain directions of the gaze,

and so appear to be pseudo-nystagmus. The old terms "nystagmus" and "pseudo-nystagmus" he would like to see abolished and "with field central," "with field peripheral," etc., substituted. If the nystagmus was jerky with a widespread field and was not changed by occlusion, it was probable that the patient had a more widespread condition. The movements in the two eyes were dissociated, probably the lesion was comparatively peripheral; whereas if it were associated, a lesion of higher and more remote centers would be suspected, one which affected the neuro-muscular end-organ. He discussed nystagmus as a neurological problem in some detail.

Dr. Hugh E. Jones (Liverpool) exhibited a model to remind one of the semicircular canals and the relation in space of these to other structures in various postures of the head. He also rotated in the chair a man who had destruction of the labyrinth on both sides following influenza, and proved the absence of nystagmus after that procedure. He subjected a normal person to the same amount of rotation and demonstrated the ensuing nystagmus.

Dr. Gordon Holmes said that in a discussion on nystagmus all the components must be taken into account, but the neurologist was mainly interested in the central mechanisms brought into play in adjusting the eyes, and in stimuli of sight in the visual field. Nystagmus could not be regarded as a mere local tremor of the eyes, it was a coördinating act. It could take place even when the muscles were palsied or paralyzed, as he had described a case in which the external rectus on the right side was palsified in association with a cerebellar lesion, yet nystagmus was regular, and symmetrical in both eyes. In nystagmus the essential feature was the slow phase. In many conditions a terminal fixation nystagmus took place, and among these were sthenic states and chronic, including alcoholic, intoxications. He had never found, in his large experience of gunshot wounds of the head in the War, nystagmus occurring from a primary lesion of the cortex or of the fore-brain as such.

Mr. Sidney Scott discussed the phenomenon from the standpoint of the otologist. He said the latter made his observations on this in cases of people with normal vision,

and he would exclude cases of central nervous disease. It was assumed that nystagmus could be set up in the labyrinth as a result of irritation, and by a caloric stimulus. The otologist was sometimes asked whether nystagmus was the cause of the vertigo, or vice versa, and the reply he gave to that was that both were due to a common cause. But nystagmus was not associated with vertigo when it was not of labyrinthine origin. In acute otitis media a nystagmus was sometimes met with which must be distinguished from that following destruction of the labyrinth. When nystagmus was due to otitis media it would disappear when the tympanum was drained and there was no immediate risk of extension of the disease to the labyrinth or to the meninges. If there was absence of response to the caloric test, the labyrinth should at once be opened. There was nystagmus also accompanying chronic fistula.

On Thursday evening Dr. Gordon Holmes gave an address on tumors involving the chiasma, not including pituitary tumors, Dr. Muloch Houwer read a paper on keratitis filamentosa and chronic arthritis, Col. H. Herbert one on glass membrane formation in irido-cyclitis. These were briefly discussed.

On Friday the scientific discussions were prefaced by the presentation by the President of the Edward Nettleship Prize for 1927 to MR. CHARLES HOWARD USHER, of Aberdeen, for his work, especially in the field of eugenics.

This session was occupied with a discussion on

The Ætiology and Treatment of Hypopyon Ulcer.

MR. J. GRAY CLEGG (Manchester) introduced the discussion, his remarks being based on 27 years as hon. ophthalmologist at Manchester Royal Eye Hospital, 160,000 patients having been under his care there in that period, 9,576 as in-patients. Of the total, 0.24% were cases of hypopyon ulcer. 77% were males. In 53% of all the hypopyon cases there was a definite history of trauma, and most of the subjects were either miners or laborers. The condition was not found in pit ponies, and it was not due to infection from underground stables, as the air currents were forced towards those

stables. Most of the patients with this condition which Mr. Clegg saw were below par in a general way. Alcoholic addiction he felt had a distinct influence in lowering resistance and in retarding recovery. In 26% of his cases of hypopyon there was conjunctivitis, and in 13% dacryocystitis. Intranasal conditions he saw only twice. It was impossible to say how far infection was brought about by workmen's dirty fingers, dirty handkerchiefs, or by attempts to lubricate the eye by sputum. The most frequent associated organism he had found in association with these cases was the encapsuled pneumococcus of Frankel-Weichselbaum.

Treatment.

Mr. Clegg stressed the importance of keeping the general health as robust as possible, by simple and nourishing diet and open air, and purgatives when necessary. Pain could be relieved by morphia or codeia. The teeth must be put right. With regard to local treatment, a bandage he considered retarded recovery as the affected region was thereby kept in contact with the pus and toxins. Among lotions he mentioned: hydrarg. per chlor. 1 in 10,000; zinc sulph. gr. j. ad. ℥1; mercurophen, 1 in 8000; quinine hydrochlor. gr. 4, ad. ℥1; formalin 1 in 2,000 or weaker. Drops referred to were: argyrol 10% to 50%; protargol 5% to 10%; dionin up to 10% strength; adrenalin 1 in 1,000. Darier, he said, strongly recommended the instillation, every two hours, of a 2% of zinc salicylate. Powders mentioned were iodoform, xeroform, nosophen, airol; and of ointments iodoform gr. xxx, ad. ℥j. A 20% ointment of cassaripe was recommended by Chandler of Boston and by Risley of Philadelphia. Subjunctival injections were usually given with one-third the quantity of acoin to overcome the pain caused. Another injection was cyanide of mercury 1 in 4,000, and another was sodium chloride 1% to 5%. To the ulcer direct could be applied hydrarg. per chlor. in solutions up to 1 in 100. The burning sensation could be overcome by instilling drops of holocain.

Heat could be applied indirectly, i.e., to the closed lids. either by the Maddox pad or by lint repeatedly wrung out in hot thin starch water. Or heat could be applied direct to

the ulcer by holding a cautery knob or loop close to the ulcerated area several times daily, to dry its surface. Or a nicer regulation of the temperature used was possible by Shahan's electrothermophore. Blue light did not prove useful. Lawson and McKenzie had advocated radium being used.

In all cases it was well to irrigate the ulcer, drying it with blotting paper, gently mopping out with a dry swab, then scraping and cutting away the projecting margin. A favorite agent to use was carbolic acid liquefactum. An accurate application was by Paquelin's benzol vapor cautery. The operative measures were entered into in detail. Of indirect measures some surgeons preferred a large peripheral section. Iridectomy was not performed. In regard to direct means, frequently scraping of the necrotic mass from the ulcer surface, especially at the advancing edge, was done. Verhoeff made crucial incisions into the base of the ulcer. A more heroic measure was the Guthrie-Saemisch section, which resulted in a lowered tension, and a slow drainage of the anterior chamber, allowing the hypopyon to be effectively dealt with. No sudden lowering of intra-ocular tension must be brought about, or hæmorrhage might result.

He also dealt at length with the bacteriology and the complications, and concluded by saying that workers could not have it too firmly impressed upon them that it was necessary to look upon a watery or discharging eye as a serious matter, and that skilled advice should at once be sought. Much protection could be afforded if men would wear goggles at their work.

Mr. S. H. Browning dealt with the subject as a bacteriologist. He said the organism most frequently associated with hypopyon ulcer was the pneumococcus, but it only became established on the cornea when its surface had become eroded. He believed infection was brought about by organisms which were already on or in the conjunctival sac and which the erosion had enabled to gain access. Cultures should always be taken from the advancing edge of the ulcer. If bacteriological examination of all ulcers were carried out, many eyes might be saved by it. The most virulent cases he had seen had been due to B. pyocyaneus, and in some of those cases

he had been able to trace the infection to an ear discharge. He was not impressed by autogenous vaccines in these conditions. He spoke of the treatment of these ulcers by magnesium sulphate and hexamine.

MR. M. S. MAYOU showed a slide of a very early stage of pneumococcal corneal ulcer following the entry of a foreign body. He treated the ulcer with pure carbolic, and it did not spread.

MR. MCMULLEN considered these ulcers were now not so frequently met with as 25 years ago. As most of the cases were preventable, prophylaxis should have first consideration. He insisted that, even for slight injuries, the treatment should be skilled. It was now his custom to use the galvano-cautery for all hypopyon ulcer cases, except in those with only slight ulceration.

MR. A. C. HUDSON said intraocular pressure exerted a very unfavorable influence on the course of the disease; it interfered with the normal lymph supply of the cornea. When the cautery was applied, it should be limited to the crescentic edge of the ulcer, and it should not be applied deeply. In addition he had used, alternatively, ½ gr. chloride of zinc drops, or 5% protargol. If Guthrie-Saemisch section were decided on, it should be done early, as tension did not need long to become seriously greater.

DR. FREELAND FERGUS said he had seen a very large number of these cases, as he had always practised in a large industrial area in Glasgow. If the ulcer was seen early, the whole treatment resolved itself into a question of asepticity; the free use of the douche and the instillation of a few drops of pilocarpine. He was against the use of both ointments and bandages, as they harbored and retained what it should be the effort to evacuate.

MR. EDGAR STEVENSON agreed with what Mr. McMullen and Dr. Fergus said. At hospitals removal of foreign bodies was usually well done, but the after-treatment of the case was practically neglected. Nurses and others brought into contact with these cases should have been well trained in eye work. The ulcers seen in coal-mining districts seemed to be specially virulent; he did not know whether this fact had any actual relation to attributes of the coal.

MR. H. MCNABB said there was no panacea for these cases. They were due to injury and infection of the lacrymal sac. Vaccines and serum therapy had not been beneficial in his hands. He always injected drops of a 20% solution of argyrol, and if dacryocystitis was present he excised the sac.

COL. MCCALLAN pointed out that most subjects of hypopyon ulcer had dental sepsis, and until this was cleared up, local measures directed to the eye were not likely to avail. Also, a septic nose should be dealt with. House-surgeons should not be allowed to carry out cauterization of these ulcers without instructions and supervision.

MR. M. HEPBURN said there was some divergence of view as to the best means of treating these ulcers. There seemed to be no means of determining, in a given case, whether an ulcer would be severe or not, therefore it was safer to adopt measures best suited to the severe condition, especially as much of the resultant scar became absorbed later. He always allowed the affected eye to remain uncovered, carrying out irrigation as often as possible. Rapid destruction of corneal nerve endings gave relief from pain, and this sometimes resulted in patients seeking advice too late. Sometimes tension was present without pain, and the relief of this was brought about by a slow evacuation of the anterior chamber. The purulent material should not be pulled out forcibly by means of forceps. He advocated an incision through the base of the ulcer.

COL. H. HERBERT spoke from a large Indian experience in cases of this sort which had been neglected before he saw them. It was important, as a primary measure, to clear the conjunctiva of organisms, preferably by a daily washing out with perchloride. He also favored cauterization of the ulcer, especially the margins.

MR. CHRISTIE REID said he had given up the use of the cautery for these cases. If pure carbolic did not cause healing, he did section. He pleaded that these cases ought to be incised earlier than was usual.

MAJOR R. E. WRIGHT spoke from a large experience of these cases in India. He considered that tension was the key to the riddle of treatment; for in adults ulcus serpens did not respond to treatment unless tension was controlled. In ex-

tensive cases he preferred the use of pure carbolic. Intravenous use of iodine was useful in his hands.

Mr. A. L. Whitehead (Leeds) said that in a large practice among industrial cases he found there were about five cases in males to one female. To the more superficial cases pure carbolic was applied, and for the deeper ones, the galvanocautery. The associated conjunctivitis was treated with 10% to 25% argyrol drops. He urged the necessity of getting some form of eye protection insisted on, as the total number of these cases was serious, both from the standpoint of health and economically.

Sir Arnold Lawson deprecated the use of salts of zinc. He favored frequent and thorough irrigation. He preferred the electric cautery to carbolic. Hypopyon ulcer in children was more amenable to treatment than the condition in adults.

Mr. J. Rowan (Glasgow) favored using a dull cautery direct to the ulcer, followed by paracentesis. He agreed as to the seriousness of these cases in coal miners.

Dr. Hugh Jones spoke of the plan of entering a Graefe knife at the limbus, and extending it until it just overlapped the margin of the ulcer. It was devised by Mr. Richard Williams.

At the concluding session on Saturday morning the following papers were read and discussed:

Mr. M. S. Mayou: "Distension of the Neural Canal in the Embryo in relation to Microphthalmos."

Miss Ida C. Mann: "The Nature and Boundaries of the Embryonic and Adult Vitreous Humor."

Mr. Frank Juler: "A Case of Lid Sarcoma with Secondary Growth, Apparently Cured by Radium."

Mr. T. C. Summers: "A Case of Papillœdema in a Pedigree Bull."

Mr. Bernard Chavasse organized a museum, which was open during the sittings of the Congress, and members were provided with a generous social and recreative programme.

REPORT OF THE SECTION ON OPHTHALMOLOGY, NEW YORK ACADEMY OF MEDICINE.

By Dr. THOMAS HAYES CURTIN, Secretary.

MEETING APRIL 18, 1927. DR. CLYDE E. McDANNALD, CHAIRMAN

Dr. Arthur Yudkin presented a case of **Bilateral prepapillary vascular loop of retinal artery** (published in this issue.)

Dr. Daniel B. Kirby read the first paper of the evening, entitled, **The cultivation of lens epithelium** (published in full).

Discussion: Dr. Davis. In the first place, I wish to congratulate Dr. Kirby on a most excellent work he has accomplished in the cultivation of the lens epithelium in pure culture. At the suggestion of Dr. Wheeler, and following in the footsteps of Drs. Carrel and Eberling, he has succeeded in producing and reproducing pure cultures of the lens epithelium. His brilliant idea of freeing these delicate cells from all extraneous matter and from all other cells brought him the great success for which he was trying, and this is one step further forward in solving this difficult problem of the nutrition of the lens—and upon that feature, nutrition of the individual lens cell, depends in a great measure, the problem of prevention of cataract.

I had the pleasure of seeing Dr. Kirby work in his laboratory, and no one who has not witnessed the extremely delicate technique of this work can appreciate the skill and judgment that is necessary to carry it through.

Granting all this, I do not think it wise to defer the treatment of cataract until all the scientific points in its solution are solved. In fact, it is often the empirical that helps to solve medical problems and to bring about cure of disease, though we do not know just how the remedies act. For ex-

ample, witness the marvelous efforts of non-specific protein therapy, about the nature of their action we know next to nothing.

About the specific proteins we know something more, and it behooves us in the meantime in combating cataract to use whatever remedies we have until we have something better at our hand.

DR. FRIDENBERG: Dr. Kirby's studies represent undoubtly a great step forward in our preliminary investigation, from the biological standpoint, of the development of cataract. The cultivation of transplants of lens epithelium and other lens tissue elements will make it possible to control some of the factors in experimentation. There is just one point to which attention might be brought, and that is the great variety of causes of cataract. Studies of lens epithelium, transplants and cultivation, and the immunological factors, represent the biological approach, but lenticular opacity, progressing to complete cataract may be due to the most varied causes. I need only mention radiant heat, as in glass-blowers' and stokers' cataract, cataract from lightning and electric discharge, cataract from the ingestion of various poisons, naphthalin cataract, and finally traumatic cataract in injuries to the eye by blunt violence where a lesion of the lens epithelium of a purely mechanical nature allows the entrance of aqueous under the capsule and produces a progressive opacification of the lens. The cataract of tetany is of particular interest in this connection as there seems to be mechanical as well as biological (endocrine, avitamine) factors concerned in its development.

Dr. Kirby, in closing. We have not been able to reproduce, with our experiment, the same conditions as in the human body.

The second paper of the evening, **The treatment of the commoner syphilitic lesions of the eye,** was read by Dr. Joseph Gardner Hopkins (published in full).

DISCUSSION: DR. A. BENSON CANNON. The most important thing is not the type of drug but continuous treatment with an efficient drug. In cases of optic atrophy, the only thing to do is to treat them with an anti-syphilitic cure. He quoted a case he saw in 1917, a man with 4 plus Wassermann

and optic atrophy. He was given five intra-spinal injections and also intra-muscular injections of salvarsan. His vision decreased until he became entirely blind. Paretics can be cured if seen early and given intra-spinal treatment. The progress of optic atrophy can be arrested by intra-spinal treatment. The patient should be advised, notwithstanding the loss of vision, to continue his anti-syphilitic treatment. The cure of a few cases justifies the treatment. Iodide alone will clear up an interstitial keratitis. We have a number of cases of interstitial keratitis whose Wassermann becomes negative with constant treatment.

DR. EGGSTON: He recited the procedure employed in the treatment and follow-up of syphilitic lesions of the eye at the Manhattan Eye, Ear, Nose and Throat Hospital. Systematic treatment of syphilis was instituted there five years ago. The patient receives 12 injections of mercury and 6 weekly injections of neo-salvarsan, followed by a two weeks' period of rest and then a Wassermann test. If the test is positive, another series of treatment is given. This is repeated until thirty-six salvarsan and seventy-two mercury injections are given, provided the patient does not become negative. If no improvement is found in the Wassermann, the treatment is shifted to bismuth. Frequently, cases that do not respond to salvarsan and mercury will respond to bismuth. After a negative reaction, the blood is retested in two months; if it is then negative, another test is made in six months and subsequently one each twelve months.

Of the 1200–1300 new cases treated at the hospital, 60% were eye cases of the various types. About two hundred new cases are treated yearly, some having had previous treatment. We rarely see a primary case, all appear to be secondary or tertiary cases. Therefore, the cases are of unknown duration and very difficult to cure. About 20% of all the cases treated have shown from one to three negative Wassermann reactions, and most of this number have shown three successive negative reactions. About 10% of all eye cases show negative reactions after extensive treatment. About 45% continue to show strongly positive reactions after intensive treatment. The remaining number show reduction in the test from 4 plus to three, two or one plus reactions.

Silver salvarsan may be of some use when other products have failed to produce a negative reaction. Thorough treatment is the most important point in any form of treatment. Neuro-syphilis should be treated intra-spinally. The technique is a little difficult, but should be insisted upon, especially in the early cases of any forms of neuro-syphilis.

Treatment of patients with interstitial keratitis should be prolonged and thorough, both to prevent recurrent attacks and for the general improvement of the patient. Two of our cases, however, have shown recurrent acute attacks in the other eye during treatment. However, these attacks have been of short duration. All cases of syphilis and especially keratitis should receive very thorough and extensive treatment.

DR. KNAPP: The two conditions that are the most interesting are; 1: **Interstitial Keratitis**. 2: **Optic Atrophy.** He is glad to hear that Dr. Hopkins is in favor of intensive treatment of optic atrophy by the intra-spinous method. There are some cases where treatment has done harm. Can one decide on the treatment by the findings of the intra-spinal fluid? It is curious that cases of iritis do not develop optic atrophy. He finds that in interstitial keratitis, notwithstanding treatment the second eye has become affected, and that this infection in the second eye is not necessarily of a milder character.

DR. COHEN: In the treatment of interstitial keratitis of luetic origin, salvarsan injection is not free from a severe local reaction. I have known this reaction to cause disastrous results to the cornea. My procedure in these cases is the usual local treatment, associated with the use of mercury and iodides.

The pallor of the disk does not always indicate an optic atrophy in cases of lues, as this appearance might be due to a luetic endarteritis. The improvement in vision and change in color of the disk indicates a luetic vascular basis and not a degenerate change in the optic nerve fibres.

DR. HOPKINS, in closing. Positive reaction of the spinal fluid indicates intra-spinal treatment. Most cases with good results are due to intra-spinal treatment. Vascular meningitis cases do well. Cases with restriction of fields with good central vision are unfavorable cases for intra-spinal

treatment. In regard to the question of iritis and optic atrophy, I have nothing to say except that cases that give marked reaction in the skin do not get optic atrophy. Those that develop early lesions get early treatment, that might account for a percentage of these cases not developing. In treatment of the keratitis cases, I find that salvarsan with iodides are most efficient.

MEETING MAY 16TH, 1927.

DR. HIDEYO NOGUCHI (by invitation), read the first paper of the evening, entitled, **Chronic granular conjunctivitis in macacus rhesus and chimpanzee, induced by a microörganism isolated from cases of American Indian trachoma.** This lecture was illustrated by lantern slides. This paper is published in this issue.

DISCUSSION: Drs. George E. de Schweinitz, Philadelphia, William H. Wilmer, Washington (by invitation), F. H. Verhoeff, Boston (by invitation), F. I. Proctor, Boston (by invitation), P. Richards, Fort Defiance, Arizona (by invitation) and Ida A. Bengtson, U. S. Public Health Service, Rolla, Missouri (by invitation). Dr. Noguchi closed the discussion.

The second paper was read by DR. HENNING RÖNNE, Copenhagen (by invitation). It was entitled **Theory and technique of the squint operation.** Due to the lateness of the hour, there was no discussion on the paper. Published in full in this issue.

EXECUTIVE SESSION: The following officers were elected for the year 1927–1928: Chairman, Dr. Bernard Samuels, Secretary, Dr. Thomas H. Johnson.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M.D., F. A. C. S., New Rochelle, N. Y.

(*Continued from July issue.*)

XI. THE UVEAL TRACT.

188. Bielschowsky, A. **Convergence immobility of the pupil.** *Klin. Monatsbl. f. Augenheilkunde*, 1923.

189. Brownlie and Neame. **A case of implantation cyst of the iris and ciliary body.** *British Journal of Ophthalmology*, vii., November.

190. Butler, Harrison, and Assinder. **A case of endothelioma of the ciliary body.** *Ibid.*, vii., December.

190a. Clapp, C. A. **Metastatic carcinoma of the choroid with report of two cases, four eyes.** *Amer. Jour. of Ophth.*, ix., 513, 1926.

191. Dwyer, Hugh L. **Aniridia in five generations.** *Journal of the American Medical Association*, lxxxiii., p. 1587.

192. Fuchs, A. **Anatomy of inversion and recession of the iris.** *American Journal of Ophthalmology*, August, 1925, p. 601.

193. Holth and Berner. **Congenital miosis or pinhole pupils owing to developmental faults of the dilatator muscle.** *British Journal of Ophthalmology*, vii., September.

193a. Irons, E. E., and Brown, E. V. L. **Recurrence of iritis as influenced by the removal of infections.** *J. A. M. A.*, lxxxvii, 1167, 1926.

193b. Jackson, Edward. **Retinoblastoma and recent views of malignancy.** *J. A. M. A.*, lxxxvii., 1471, 1926.

194. Karpow. **Myotonic accommodation and pupillary reaction.** *Vereinig. Wuerttemb. Augenaerzte*, May 10, 1923.

195. Kestermann, Gertrud. **Immobility of the pupil to accommodation without immobility to light.** *Klin. Monatsbl. f. Augenheilkunde*, lxx., 1923.

195a. Key, Ben Witt. **Sympathetic ophthalmia cured after exenteration of the nasal accessory sinuses.** *Amer. Jour. of Ophth.*, ix., 574, 1926.

196. KNIGHT, MARY S. **A critical survey of neoplasms of the choroid.** *American Journal of Ophthalmology*, October, 1925, p. 791.

197. LAGRANGE. **Pneumococcal panophthalmia and pneumococci in the blood.** *Annales d'oculistique*, clx., 8, p. 626.

197a. LENT, E. J. and LYON, M. B. **Hemangioma of the choroid.** *Amer. Jour. of Ophth.*, ix., 804, 1926.

198. LICZKO, A. **A rare case of atrophy of the iris.** Hungarian Ophthalmological Society, November 5, 1923.

199. MEISSNER. **A peculiar threadlike formation in the anterior chamber.** Ophthalmological Society in Vienna, April 23, 1923.

200. MEISSNER. **Diagnosis of sarcoma of the choroid.** *Klin. Monatsbl. f. Augenheilkunde*, May-June, 1923, p. 722.

201. PROKSCH. **A case of melanoma of the papilla and choroid.** *Zeitschr. f. Augenheilkunde*, li.

202. REMKY, E. **Spontaneous cysts of the posterior surface of the iris and of the ciliary body.** *Klin. Monatsbl. f. Augenheilkunde*, lxx., 1923.

202a. SAMUEL, E. C. and BLUM, H. N. **Roentgenotherapy of exudative iritis.** *J. A. M. A.*, lxxxvii., 1033, 1926.

202b. RODIN, FRANK, H. **Treatment of iridocyclitis by subconjunctival injections of atropin and epinephrin.** *Amer. Jour. of Ophth.*, ix., p. 24, 1926.

203. SCHIECK. **The uvea in retinitis albuminurica.** *Ibid.*, lxix., 1922.

203a. SCHWARTZ, F. O. **Melanosarcoma of choroid, sympathetic ophthalmia and retrobulbar neuritis.** *Amer. Jour. Ophth.*, x., p. 35, 1927.

203b. SHAHAN, W. E. **Panophthalmitis, evisceration, sympathetic ophthalmia.** *Amer. Jour. Ophth.*, x., 120, 1927.

204. USHER. **Cases of metastatic carcinoma of the choroid and iris.** *British Journal of Ophthalmology*, January, 1923.

204a. ZENTMAYER, WILLIAM. **The prostate as a remote focus of infection in ocular inflammation.** *J. A. M. A.*, lxxxvii., 1172, 1926.

DWYER (191, **Aniridia in five generations**) encountered fifteen cases in five generations. The facial expression of the children is characteristic, the palpebral fissure being voluntarily narrowed and the chin held down on the chest for they seem to suffer from photophobia. Nystagmus is marked. The adults show corneal lesions also. ALLING.

LICZKO (198, **A rare case of atrophy of the iris**) reports the case of a man 53 years old who had noticed a hole in his right iris fifteen years before. At the time of examination the tension of the eye was elevated, and the temporal third of the iris was wanting. An iridectomy at another place did not reduce the tension. Vision became lost, there was degeneration of the cornea and atrophy of the iris. The Wassermann reaction was three plus. The diagnosis was

primary syphilitic atrophy of the iris with secondary glaucoma.

MEISSNER'S (199, **A peculiar threadlike formation in the anterior chamber**) patient stated that during a febrile attack three weeks before he had noticed that he could not see well with his left eye. Nothing was known of any previous eye trouble, and the nature of the febrile disturbance remained uncertain. The eyeball and its adnexa were free from inflammation or irritation. An opacity in the outer, lower quadrant of the cornea resembled the degeneration of keratitis vesiculosa. The iris seemed to be of normal structure, yet at the lower angle of the chamber was an aperture, looking like a black spot. In the anterior chamber a bright, threadlike formation was visible, starting from the posterior surface of the cornea, having a knoblike thickening in the middle, and passing downward and outward over the iris in a long drawn out S curve to disappear in the angle of the anterior chamber. On the temporal side of the aperture in the iris a second, thinner thread branched off and ran upwards, but had no connection with the iris. Under the corneal microscope the formation appeared to be structureless with brownish deposits at certain places. The condition remained unchanged while the patient was under observation. The writer does not think that he had to deal here with remains of a pupillary membrane because there was no connection with the iris and no iritic structure.

FUCHS (192, **Anatomy of inversion and recession of the iris**) describes a lesion of inversion of the iris as lying completely back upon the ciliary body and recession where the iris is folded and incarcerated between the ciliary body and lens. Clinically in cases of inversion nothing can be seen of the iris at the point of inversion, whereas in recession it is greatly narrowed at this point. He supposes that recession represents a slight degree of inversion and is often a transitional stage between an inverted iris and a normally placed iris. ALLING.

An advancement of the external rectus had been performed on a boy 10 years old. Eight months later he came under the observation of BROWNLIE and NEAME (189, **A case of implantation cyst of the iris and ciliary body**) who found a

perfectly transparent cyst on the posterior surface of the cornea in the outer and lower angle of the anterior chamber. The conjunctiva at this place was cicatricial and thickened, except for two round places through which the ciliary body appeared black. Two attempts to extirpate the cyst failed, each time there was a recurrence. Histological examination showed that the cyst was located in the anterior part of the ciliary body and in the root of the iris. It was completely lined with epithelial cells, and ciliary body tissue with some pigment cells was to be seen on its posterior wall. Apparently one of the stitches was placed too deeply during the advancement, wounding the ciliary body, tearing and implanting the conjunctiva.

REMKY (202, **Spontaneous cysts of the posterior surface of the iris and of the ciliary body**) observed an immature cataract in a man 62 years old. When the patient returned, six months later, he found in the other eye, which was not cataractous, a black brown tumor behind the iris which was opaque and pressed the iris forward into the anterior chamber. The diagnosis of sarcoma was made and the eye enucleated. But the pathological examination showed the tumor to be a multilocular cyst on the posterior surface of the iris.

HOLTH and BERNER (193, **Congenital miosis or pinhole pupils owing to developmental faults of the dilatator muscle**) observed this condition in two sisters and a brother. No examination of the eyes of the parents was possible. All of the six pupils had a diameter of about 0.5*mm*, and could be dilated to 2.5*mm* by atropine or scopolamine. The iris showed no contraction furrows, no posterior synechiæ, and there were no traces of persistent pupillary membrane. One patient had a marked spasm of accommodation which caused severe headaches. She was 47 years old and for 26 years had used atropine or scopolamine for the relief of the headache, when she had $\frac{5}{8}$ vision with a weak concave glass, although in youth she had worn -10 or -12 D. The other two patients had no such trouble. One was highly myopic, the other emmetropic. Both of these patients died of apoplexy at the ages of 56 and 57. The histological examination of their eyes gave in general the same condition, except for differences of degree. The sphincter was very strongly developed,

but the dilatator very imperfect. The posterior surface of the iris in the area from which the dilatator was absent had an almost smooth appearance, the stroma was less dense and partook of the character of fœtal, mucous, or gelatinous tissue.

KARPOW'S (194, **Myotonic accommodation and pupillary reaction**) 20-year-old patient, who had been 11 years under observation, formerly had a right-sided mydriasis with weak reaction to light and convergence without involvement of the accommodation. At this time she had mydriasis of the one eye with a slightly distorted pupil and otherwise normal iris. The reaction to convergence was very marked and lasted ten seconds; the return to dilatation lasted twenty seconds. The reaction to light was of a tonic character and not marked. There was no unrest of the pupil, no dilatation in response to sensory irritation, no orbicularis phenomenon. The accommodation had a normal range; it required five seconds to fix upon the near point, likewise five seconds to return to a state of rest. No change in this was secured by having the patient repeatedly look at distance and near in alternation. No cause could be determined other than a distinctly neuropathic condition of the patient.

KESTERMANN (195, **Immobility of the pupil to accommodation without immobility to light**) discusses the case of a man 64 years old, who had been for nineteen years under the observation of Prof. O. Schwartz of Leipsic. There was no history of syphilis and the Wassermann test had always proved negative, yet at first the accommodation had improved somewhat under specific treatment. The presence of a pathological focus in the region of the oculomotor nucleus was considered possible, while syphilis as a cause was thought to be doubtful.

BIELSCHOWSKY (188, **Convergence immobility of the pupil**) says that in twenty-five years experience with an abundance of material he had never seen a single case of pure convergence immobility, so he was inclined to believe such cases to be very rare, and that diagnostic errors had been made in some of those reported. Now he has seen his first case in a woman 21 years old, and he believes the condition to have been congenital.

By means of an irradiation of X-ray of less than one-twentieth of an erythema dose through a millimeter of aluminum plate, SAMUEL and BLUM (202a, **Roentgenotherapy of exudative iritis**) found favorable response in eleven cases which they treated. They suggest that this procedure should be adopted in all stubborn cases of exudative type of iritis.

YUDKIN.

ZENTMAYER (204a, **The prostate as a remote focus of infection in ocular inflammation**) reports four cases in which ocular tissue is affected. He notes that the metastasis, as in other focal infections, may occur in any of the ocular tissues susceptible to inflammatory reaction, but the uvea and cornea are probably most often involved; the latter and the iris are especially injured when the gonococcus is the invading organism. YUDKIN.

Continuing their study on a new group of 50 cases, IRONS and BROWN (193a, **Recurrence of iritis as influenced by the removal of infections**) found that in 43 cases there was no recurrence of iritis after the removal or adequate treatment of infections found and believed to be the cause of the iritis. In four others in which there were recurrences, the infections were known not to have been removed. The remaining cases had complications which were difficult to overcome. The series emphasizes the importance of thorough examination and removal of infectious processes in iritis. YUDKIN.

RODIN (202b, **Treatment of iridocyclitis by subconjunctival injections of atropin and epinephrin**) found that in many instances a combined subconjunctival injection of atropin and epinephrin dilated the pupil to the maximum in acute iridocyclitis when instillations of four percent atropin had failed. An injection of two minims of 2% atropin and four minims of epinephrin 1:1000, gave rapid or instantaneous dilatations of the pupil in some cases. The combination of drugs had little or no effect on old adhesions; age is no contra-indication to the injection.

YUDKIN.

BUTLER, HARRISON, and ASSINDER (190, **A case of endothelioma of the ciliary body**) report the following case. A man 55 years old had complained of failing vision in his right eye for five years. The tension of the eye was normal.

In the upper, temporal portion of the iris was a yellowish white tumor with little pigment which threw a shadow on transillumination. In addition there were posterior synechiæ and cataract. The histological examination of the enucleated eye showed the tumor to be an endothelioma of the ciliary body instead of a sarcoma as had been thought. It was composed of large, irregularly margined cells with large nuclei and abundant vacuoles, probably coming from the lymph spaces of the region of the ciliary body. The pigment deposits came secondarily from the posterior surface of the iris.

KNIGHT (196, **A critical survey of neoplasms of the choroid**) found an example of hemangioma and plexiform neuroma. These types of choroidal tumors are rare. The hemangioma involved the choroid around the posterior pole and circled the optic nerve. The growth did not show signs of malignancy. In all the cases reviewed from the literature the growth was limited to choroid and was strictly a developmental anomaly rather than a true neoplasm. The eye showing neurofibromatosis microscopically was a typical example of buphthalmos. Although it was not associated with neurofibromatosis about the orbit or elsewhere the choroid showed the same structure as has been described and recorded several times in the literature as neurofibroma. This lesion in the choroid can also be grouped with the developmental anomalies for it showed none of the characteristics of malignancy. The melanomas represent the sole type of primary malignant tumors of the choroid. ALLING.

LAGRANGE (197, **Pneumococcal panophthalmia and pneumococci in the blood**) reports three cases of panophthalmitis in which the active agents were pneumococci, and in which cultures demonstrated the presence of pneumococci in the blood. In the first case the panophthalmitis was metastatic during an attack of grippe with involvement of the lungs, in the second it followed perforation of an ulcer of the cornea, in the third it resulted from a perforating wound. In the second and third cases the pneumococci may have entered the circulation from the wound in the eyeball.

When it is difficult to determine whether a case is one of sarcoma of the choroid, or a simple detachment of the retina, MEISSNER (200, **Diagnosis of sarcoma of the choroid**) aspirates

the detachment and examines the fluid microscopically. If bands of cells are found, they are looked upon as showing the presence of a tumor, while in a serous detachment only swollen pigment epithelia or single inflammatory cells are to be found. Such tumor cells Meissner found in five cases out of six, and the diagnosis was confirmed by operation in four, the fifth refusing to allow his eye to be enucleated. It is not desirable to puncture the tumor itself, but simply to aspirate the fluid behind the detached retina.

JACKSON (193b, **Retinoblastoma and recent views of malignancy**) suggests the term retinoblastoma for the poorly labelled malignancy of early childhood called glioma of the retina. The condition is congenital and is frequently bilateral. A familial tendency has been recorded by many observers. It is regarded as uniformly fatal. YUDKIN.

PROKSCH (201, **A case of melanoma of the papilla and choroid**) observed in the fundus of the right eye of a man 40 years old, a gray, fairly circular spot, of 1.5 pupillary diameter, which involved almost the entire lower half of the papilla and the neighboring tissues. It lay behind the retinal vessels and nerve fibers and formed neither a protrusion nor a depression. It was plainly not malignant as the function of the eye was undisturbed and the condition did not change within six months. He considered it to be a benign melanoma, such as hitherto has been met with only in the periphery.

From a critical study, LENT and LYON (197a, **Hemangioma of the choroid**) found the lesion to be a rare pathological condition. It was most frequently observed during the second decade. It did not appear to grow rapidly. Detachment of the retina and glaucoma was the ultimate result. They report a case of this type with histological findings.

YUDKIN.

The first case described by USHER (204, **Cases of metastatic carcinoma of the choroid and iris**) was that of a man 36 years old, who had a failure of vision in his left eye. No positive diagnosis could be made at first from the uncharacteristic swelling of the retina, but soon a detachment appeared in the lower half and the eye was enucleated because the presence of a tumor was suspected. The tension of the eye did not seem to be elevated on palpation. Sometime later the pa-

tient died with obscure clinical symptoms. Unfortunately no autopsy could be secured. The histological examination of the eye revealed a papillary alveolar carcinoma of the choroid. The detachment of the retina beneath the tumor was produced by an exudate. The second case was that of a woman 35 years old from whom a mammary carcinoma in an advanced stage had been removed. A short time after the operation both eyes became blind. Detachment of the retina in both eyes, though the diagnosis as regards the right eye was uncertain at first. The tension did not seem raised to the fingers. Death a year after the operation. Each eye contained an extensive flat carcinoma of the choroid, which reached to the ora serrata, had infiltrated the sclera, and broken through into the papillary portion of the optic nerve. The third case was one of carcinoma in the choroid and iris in a man 48 years old. He suffered first from a cystic growth in the left iris and an uncertain gray protrusion forward of the retina without elevation of the tension. The eye was enucleated as probably containing a tumor. Shortly afterwards detachment of the retina took place in the right eye. The patient died a short time after from an attack of recurrent paralysis. Autopsy revealed a primary carcinoma of the thyroid with metastases in the liver, kidneys, and lungs.

From a critical study, CLAPP (190a, **Metastatic carcinoma of the choroid with report of two cases, four eyes**) found that the average duration of life, after the discovery of the growth in the eye, was eight months, although death occurred in one month and as late as two years. The metastasis was most frequent after breast involvement and the left eye seemed to be the first affected. One third of the cases occurred bilaterally. YUDKIN.

SCHIECK (203, **The uvea in retinitis albuminurica**) says that recently the attempt has been made to adduce from the accumulations of wandering cells in the uvea proof that these, as well as subretinal effusions, are not the consequences of stasis, but of an inflammatory œdema of toxic origin. But if one examines the anterior part of the uvea he will see with relative frequency large, wide branched spaces in the tissue of the iris. The stroma has been pressed apart by a large œdema. The sections of the vessels lie like islands in the

areas soaked with fluid. Yet no trace of any inflammatory change can be found. These changes in the anterior part of the uvea must be due wholly to an œdema from stasis, and from this, conclusions may be drawn as to the nature of the changes in the posterior segment of the uvea.

KEY (195a, **Sympathetic ophthalmia cured after exenteration of the nasal accessory sinuses**) describes a case which from a clinical and pathological standpoint was diagnosed as true sympathetic ophthalmia. The removal of the injured eyeball did not alleviate the process in the other eye. The vision in the sympathizing eye was 5/200. The nasal accessory sinuses which were found to be infected were drained and the vision in the latter eye was finally improved to 20/20. The author believed the uveitis in both eyes was due to a focus of infection located in the nasal accessory sinuses. From this observation he deemed it reasonable to assume that sympathetic ophthalmia was a specific type of uveitis due to a focus of infection or systemic disease, either exogenous or endogenous, and that the clinical picture and the destructive nature of the sympathetic type were due to pigment hypersensitiveness and hypersusceptibility of both eyes, incident to uveal destructive changes from injury to the globe.

YUDKIN.

SCHWARTZ (203a, **Melanosarcoma of choroid, sympathetic ophthalmia and retrobulbar neuritis**) reports a case of melanosarcoma of the choroid in the right eye which was followed by a sympathetic ophthalmia in the left eye. The condition in the sympathizing eye did not clear by removal of the ocular disturbance. After the tonsils were removed and the sinuses drained, the inflammation subsided and the vision was restored. He believes that sympathetic ophthalmia depends not so much upon the injury of the sympathogenic eye itself for its development, but its manifestations are due to some initial process remote from either eye, but directly influencing it.

YUDKIN.

SHAHAN (203b, **Panophthalmitis, evisceration, sympathetic ophthalmia**) reports a case of injury to an eye which was followed by panophthalmitis. The eye was eviscerated but sympathetic ophthalmia developed in the other one. The

complete removal of the original eye did not alleviate the condition. Extensive salicylate therapy relieved the sympathetic ophthalmia with restoration of normal vision.

YUDKIN.

(*To be continued.*)

BOOK REVIEWS.

IX.—**Die Pathogenen Microorganismen des Auges.** (The Pathogenic Microörganisms of the Eye.) By PROF. Dr. F. v. HERRENSCHWAND, Innsbruck, 310 pages, with 17 illustrations in the text, 12 coloured plates and one table. Published by Urban & Schwarzenberg, Berlin and Vienna, 1927. Price Mk. 24.

This book is an endeavour to enable the ophthalmologist without special bacteriological knowledge to take the first steps in the identification of the organisms in question. Each organism which may be pathogenic to the eye is described, with its occurrence in other diseases, its animal inoculation, and finally, the eye diseases it causes. A bibliography to include the year 1926 is appended. There is an introductory chapter on the general morphological and biological peculiarities of pathogenic microörganisms. The technique of obtaining the specimens in the case of eye diseases, their coloration, growth, animal inoculation are carefully gone into, so that even a beginner will find the bacteriological examination in eye diseases simplified. The more elaborate immunological and serological problems are necessarily omitted. On 12 plates drawings in colour illustrate the usual microörganisms found in ocular lesions.

The author has admirably succeeded in his undertaking and the book will be essential to anyone who has to deal with bacteriological questions. A great deal of information has been judiciously collected with great labour by one who is recognized as an authority in this field. The author is to be complimented on making this often intricate material available in a form which will not dismay the ophthalmologist and should lead to greater interest in this often neglected field.

For those who desire to pursue their studies still further the bibliography will be found of great use.

A. K.

X. **Tuberculose de l'Oeil et de ses Annexes.** (Tuberculosis of the Eye.) By DRS. E. ROLLET and A. COLRAT. 206 pages, with 24 illustrations and 4 coloured plates. Published by Gaston Doin & Cie. Paris, 1927. Bound, price Fr. 60.

As the authors state in their preface, no book has been published in French which deals exclusively with tuberculosis of the eye. In most cases ocular tuberculosis is only a manifestation of a deep infection which is habitually latent, but is of great interest, as it may be the alarm signal of and draw attention to an unrecognized danger. General treatment is, of course, of great importance, but local treatment must not be neglected though it is frequently difficult. The authors are not in favour of tuberculin therapy and have criticized the results published with this treatment in other countries, especially as they believe that there are newer therapeutic measures which are more active and surely less dangerous. The various parts of the eye are taken up in turn as they are affected by tuberculosis with a full description of the pathology, clinical course, and treatment, giving the authors' personal views based on a large experience and a review of the important publications. In discussing tuberculosis of the uveal tract the authors state that these cases are all of secondary origin and that the primary focus of infection is situated in the peribronchial lymph nodes. The X-ray has shown that there is a defined shadow situated at the base of a lobe, usually the lower lobe, surrounded by healthy parenchyma; peribronchial striæ can be traced leaving this shadow and connecting it with the hilus gland shadow. In their study of more than 100 cases the authors were able to find in at least 80% evidence of other tuberculous lesions and of definite antecedent symptoms. The proportion of cases will probably be increased by the systematic use of radioscopy in demonstrating tracheo-bronchial adenopathies. The authors' objection to tuberculin lies in the fever reactions and the difficulty of correct dosage. With certain precautions radiotherapy

is as safe as the X-ray. The technic of the application is described on pages 130–132 with a report of the results obtained. Timely enucleation of a useless and painful eye gives happy results. The authors are not convinced that tuberculosis plays a rôle in retinal and vitreous hemorrhages, nor is a cause for retinal detachment.

The illustrations in colour show experimental tuberculosis of the choroid (Rollet et Aurand) and clinical appearances of tuberculosis of the iris.

A. K.

XI.—**Die Tuberkuloesen Erkrankungen des Auges** (The Tuberculous Diseases of the Eye.) By Dr. Rudolf Bergmeister, Vienna, 144 pages, published by S. Karger, Berlin, 1927. Price Mk. 5.40.

The frequently appearing publications on ocular tuberculosis show the interest that is now taken in this subject and the prevalence of tuberculous lesions of the eye as originally advanced by von Michel is now gaining ground among ophthalmologists. Observations made in a general hospital in Vienna with many tuberculous patients led the author to review the subject of ocular tuberculosis. In a discussion of the relation of general tuberculous manifestations to ocular lesions in which the well-known views of Ranke are fully considered, it is stated that the regionary lymph nodes may remain depots for virulent bacteria for many years until they accidentally break into the blood or lymph circulation. As the general symptoms are so slight, tuberculin has attained prominence for diagnosis in ophthalmology and the intracutaneous method (Mantoux) is at present in general use. The tuberculous affections of each of the ocular tissues are then taken up in turn with a full consideration of the literature. Unusual are the observations of corneal necrosis in lupus, a tuberculogenic corneal dystrophy, pigment wandering in choroidal lesions as a result of the local reaction and cicatricial contraction of the sclera in scleritis where accumulated local reactions had been overlooked. Treatment consists in the administration of tuberculin in which the author is a firm believer; the various preparations and their application are carefully described. The value of X-ray therapy is especially

noted in the obstinate cases of tuberculous scleritis, and iridocyclitis where tuberculin treatment is often disappointing. Lymphocytosis is desirable, so is hyperemia through hot compresses; light therapy and suitable climatic conditions are helpful.

A. K.

XII.—**Diseases of the Eye.** By SIR JOHN HERBERT PARSONS, London, Fifth Edition, 676 pages, 21 plates, 343 text figures, New York, 1927. The Macmillan Co., price $6.

Parsons' excellent text book now appears in a fifth edition. The text has been revised, new matter relative to slit lamp observations has been added and some of the figures have been redrawn, while a few new ones have been added. The many splendid features of this book need not be repeated. It is scientific, yet practical, and remains the best treatise on the eye of its size in the English language, adapted not only for the student but also for the experienced practitioner.

A. K.

XIII.—**Ueber die Rhinogene Neuritis Retrobulbaris.** (Rhinogenic Retrobulbar Neuritis.) By PROF. DR. J. MELLER and DR. OSKAR HIRSCH, Vienna, 62 pages, with four illustrations in the text and two coloured plates. Published by S. Karger, Berlin. 1927. Price Mk. 4.80.

The relation of retrobulbar neuritis to diseases of the nasal accessory sinuses was thoroughly gone into at a joint session of the Ophthalmological and Rhinological Societies in Vienna in 1920 and demonstrated that many rhinologists did not share the ophthalmologists' opinion that an operation on the nasal sinuses was indicated nor even justified in cases of retrobulbar neuritis *when the nasal examination was negative.* This factor, and because the rhinologist has contributed new and valuable information on the association of these two diseases, has induced Meller, as ophthalmologist, and Hirsch, as rhinologist, to bring this subject up again for consideration in the present monograph. Mention is made of Herzog and Behr's views that the infection for the retrobulbar neuritis in multiple sclerosis takes place through the nose. The difficulty of the whole question on rhinogenic retrobulbar

neuritis lies in the fact that the diagnosis cannot be made from the ophthalmological findings alone, as the clinical signs are not characteristic. To Meller there can be no question but that retrobulbar neuritis can be caused by an occult sinusitis and that an operation in the nose must be done. In the opinion of experienced rhinologists it is the sinus cases with slight changes that cause optic neuritis and, strange to say, mild treatment frequently suffices. The inflammatory process in the nose is one which does not lead to the formation of pus.

The rhinological part is written by Dr. Oskar Hirsch. He describes the pathology of the sinus lesions and draws attention to the important investigation of Herzog, who has shown that the optic nerve is involved, not necessarily by destruction of the intervening bony wall, but by extension of an inflammation along the medullary spaces in the intact bone from the sphenoid cavity to the optic nerve sheath. Sinusitis invades the orbit through fistulæ in the bone, by way of them edullary spaces, which in places connect the nasal mucosa with the periorbita, or by way of vessels connecting the vessels of the orbit and of the nose. All three possibilities have been demonstrated at autopsy. In the investigation of the nose in these cases, discharge from these cavities is not to be expected. The microscopic examination of the ethmoidal mucous membrane shows the presence of definite inflammatory changes which continue into the bone, thus explaining the extension of disease from the nasal sinuses to the optic nerve sheath. Herzog believes that the inflammation extends along the preformed blood and lymph vessels and then through the medullary spaces in the intervening bone to the optic nerve. He regards rhinogenic optic neuritis in most cases as the result of an inflammatory œdema of the optic nerve sheath. It is well known that the catarrhal type of inflammation in the nose is characterized by great œdema. The exudation takes place into the substance of the mucous membrane and transforms it into a thick gelatinous tissue. This symptom is favorably acted upon by those means which shrink the tissues, such as cocaine and adrenalin. Finally, the intranasal operations on the ethmoidal sinus and antrum are described and some excellent illustrations appended. The author concludes:

In inflammation of the accessory nasal sinuses which extends to the optic nerve, treatment should first consist in tampons to shrink the surface of the ethmoid. If this is insufficient, the accessory sinus in question should be opened. This operation in the hands of a practised rhinologist is not dangerous.

XIV.—**Das Auge, seine Schaedigungen, ihre Verhuetung und Bekaempfung.** (The Eye, Its Injuries and Their Prevention.) By PROF. DR. VIKTOR HANKE. Vienna, 128 pages, 38 coloured illustrations. 1927, Julius Springer, Vienna. Price Mk. 4.35.

This book, written in untechnical language, is intended for teachers, parents and educators who require to know something about the eye. After an introduction on the anatomy of the eye and on the physiology of vision, the subject of blindness, its causes and their prevention, is taken up. Then follow chapters on the hygiene of the eye, on the choice of profession or trade according to their demands on vision, the diseases of the eyes which are caused by certain trades, and finally schools for the weak sighted. This is an excellent and timely book, well printed and illustrated, which reflects credit both on the author and on the publisher.

A. K.

XV.—**No. IX Die Bestimmung des Astigmatismus.** (The Determination of Astigmatism.) BY PROF. K. LINDNER, Vienna. 1927. 111 p. with 83 illustrations. S. Karger, Berlin. Price Mk. 7.50.

After giving a clear account of Landolt's explanation of the optical basis of skiascopy the author proceeds to a consideration, first, of spherical errors of refraction, and then, more fully, of astigmatism and the theoretical basis of skiascopy with cylinders. Though not original with him in concept, the systematic and thorough development of this method to the point where it is now a well recognized clinical procedure, may be regarded as Lindner's contribution. The book is a practical as well as a theoretical guide to this procedure, and to this end there are included not only detailed instructions as to technique, but also good illustrations of the optical phenomena observed in the eye under examination.

While Lindner does not discuss the relative merits of the method, it is one which is held in favor in Europe. Probably its most definite advantage is the accuracy with which it enables the position of the axis of the cylinder to be determined.

O. P.

NOTICE.

As a memorial to William MacKenzie and with a view to the encouragement of original work in Ophthalmology a "William MacKenzie Memorial Fund" has been established in Glasgow. A small committee will be elected annually, consisting of Ophthalmic Surgeons, representing the University of Glasgow, the Royal Faculty of Physicians and Surgeons of Glasgow, and others, which will have a wide discretion as to the manner in which the income of the Fund is to be expended. The "William MacKenzie Medal" will be awarded, from time to time, to the author of original work of outstanding merit in Ophthalmology, and, so far as the income permits, grants may be given in aid of research or for other kindred purposes.

Contributions towards the Fund are being received by Dr. A. J. Ballantyne, 11 Sandyford Place, Glasgow, C. 3.